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DIFFERENTIAL DIAGNOSIS

WITH

CLINICAL MEMORANDA

The
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INTRODUCTION TO

THE OUTLINES OF THE PRINCIPLES

OF

DIFFERENTIAL DIAGNOSIS

WITH

CLINICAL MEMORANDA

BY

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PREFACE

The number of works on medicine, and its various quasi-special branches, that claim the time and attention of over-burdened students and practitioners is so great that only one excuse should be offered for adding to it, viz. either to say something new or to put forward a more rational and simpler arrangement of what is old.

Very little that is new will be found in the following pages, but I claim that I have attempted to arrange the old, old phenomena of disease in such a manner as to show more clearly their fundamental meanings and relationships. I have utilised the data of physiology, and the facts of pathological anatomy, as the source from which to draw inferences and deductions, which in their turn constitute a critical analysis of clinical symptoms; I have endeavoured by this analysis to lead up to the underlying principles which govern disease as well as health. Once these principles, which are few in number, are recognised, bedside symptoms become merely illustrations of them, varied, it may be, by local and individual peculiarities, yet ever stamped with such a likeness that the simplest induction will speedily explain the organ of their origin. Isolated, or apparently isolated, facts thus lose their isolation, and become members of a related community; they no longer require separate efforts of memory for their retention, but fall naturally into their places as deductions from a universal law.

I thus hope that what I have written will serve not as a text-book of medicine, or, indeed, as a storehouse of facts, but as a series of pegs whereon to hang a chain of knowledge which will be ever increasing link by link as experience grows more ripe, and a larger and larger number of varieties of symptoms are recognised as varieties, and not elevated to the rank of species by ignorance of the connecting links.

Inasmuch as there is little here that is new, I have mentioned very few authorities for the clinical points I have utilised; they are the common possession of the medical profession at large. A few original ideas of my own are scattered throughout the work; time and clinical testing alone can show their value, but they are at least founded on some considerable experience, and the method of their exposition is that which I have found useful in teaching by the bed-side. My great wish has been to be as accurate as possible, so as not to mislead by false statements, and so sin by commission; the omissions are only too glaring, but limitation of space forbade further inclusions.

FRED. J. SMITH.

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CHAPTER I

INTRODUCTORY REMARKS ON DIAGNOSIS IN GENERAL

Dr. Pye Smith once defined pathology very pithily as "a chat about the etiology of disease." Though I think the definition somewhat too narrow, we may accept it provisionally and then define diagnosis as "the complete analysis of etiology," for we cannot consider a diagnosis as absolutely final and finished until we have probed etiology to the bottom. The process of diagnosis, thus scientifically considered, will then consist of three stages:—(1) The collecting of as complete a list as possible of the immediate phenomena of disease which are universally and by common consent termed symptoms and physical signs, *e.g.* cough, pain, diarrhœa, paralysis, palpitation, etc. (2) Inferring or deducing from these collective phenomena a primary or gross diagnosis as to the organ or organs affected and the general nature of the disease, *e.g.* bronchitis, dyspepsia, neuritis, morbus cordis, etc., thus arriving at what we may call the generic title of the affection. (3) A final step, viz. the identification of the specific cause at work, thus adding a specific name to the generic one, *e.g.* tubercular bronchitis, gouty dyspepsia, septic pneumonia, rheumatic arthritis.

The first of these three steps is naturally the simplest proceeding, for it consists only in taking a complete history of the case. Each hospital school has its own particular method of case-taking, and each teacher naturally thinks his own school the best, and I therefore give a preference to the following card, which is in use at the London Hospital. It has, I believe, at least the merit of not omitting anything of importance if used systematically.

Instructions for case-taking in Medical Wards:—

I. The Clinical Clerk to enter the heads of the case, and date of the Patient's entry to Hospital, and fill in the registered number as soon as possible. Insert dates at which the notes are taken.

II. State what the patient complains of—using his or her own words *verbatim* as far as possible, and when using the names of the days of the week, be sure to insert also the day of the month.

III. **Family History**—Number and condition of health of those living. Ages and diseases of those dead. Note any family tendency to disease.

This section is **especially** useful for the following diseases—rheumatism, gout, phthisis, hæmophilia, nervous diseases, asthma, heart disease; though it must not be omitted in any case.

Personal History—Habits (stating quantity and kind of alcohol taken), occupations, residences, previous diseases and illnesses, noting especially the dates of illnesses or changes. If in Hospital before, get the registered number, *and* **insert it in the present notes.** Especially, never omit to inquire for any disease which may stand in etiological relationship with the present illness, *e.g.* rheumatism with tonsillitis or morbus cordis, gout and lead poisoning with cirrhotic kidney, syphilis with many conditions; *these three diseases should* **never** *be omitted*, their absence, as well as presence, being equally carefully noted.

Present Illness—Date and manner of commencement: order of symptoms and dates of occurrence. A useful question here is—When were you last quite well? and trace the illness step by step from this.

Probable Cause—Insert here your own opinion and also that of your patient, and any higher available opinion.

IV. **Present Condition**—General condition. This should never be omitted; the most important items are perhaps—expression of face, colour of face (anæmia, cyanosis, etc.), œdema, nutrition, distension of veins, sleep, easy respiration, etc.; note position of patient in bed, whether sitting up or lying down, whether restless or comfortable and quiet, etc.

V. **Digestion**—Tongue, teeth, throat, appetite, vomiting, hæmatemesis, bowels.

Symptoms—Fulness or pain after food, flatulence, colic or other disturbance. Abdominal pain or tenderness.

Liver—Size and character as determined on percussion and palpation. Whether tender or not. Jaundice.

Spleen—Percussion, palpation.

Abdomen—Its physical condition as indicated by palpation, percussion and mensuration. Distension, retraction. Ascites. Tumour. Abdominal pain.

VI. **Vascular System**—**Pulse** frequency and characters, condition of arteries, veins, capillaries. **Heart** palpate, percuss, auscultate.

Symptoms—Palpitation, præcordial dulness, pain, etc.

VII. **Respiratory System**—Dyspnœa, frequency, characters of respiratory movements. **Cough**, expectoration, hæmoptysis.

Physical examination, inspection, palpation, percussion, auscultation, condition of larynx.

VIII. **Nervous System**—*General condition.* Intelligence, mental state, sleep, speech. Vertigo, delirium, coma. Complaints of head pain, vomiting, numbness, neuralgia. Paralysis, convulsions, spasms, tremor.

Motor power—Limbs, trunk, action of sphincters. Ability to walk and do ordinary work. Power over large joints, small joints, finer movements of fingers, *e.g.* writing.

Cranial nerves—Movements of eyes, tongue, face, palate. State of pupils.

Special senses—Ophthalmoscopic appearances. This should never be omitted, but is more especially important in (1) anæmia, (2) Bright's disease, (3) cerebral and spinal diseases, (4) syphilis, (5) any disease in which amblyopia is complained of.

Sensibility—Tactile sensibility of skin, anæsthesia, hyperæsthesia, dysæsthesia (perverted sensations, *e.g.* numbness, burning pain, etc.). Sensibility to heat and cold.

Reflexes—Superficial and deep. Patellar, plantar, wrist, abdominal, cremasteric, etc.

In *Spinal Cases*, gait in walking, power in each limb, condition of sphincters.

In any nervous case the special points to be attended to without exception in any case are (1) hereditary, (2) manner and time of onset, (3) condition of reflexes, (4) static and locomotor equilibration.

IX. **Locomotor System**—State of bones, muscles, joints, scars, nodes. *Skin*, moist or dry. Bed sores.

X. **Lymphatic Glands**—Neck, groins, axilla. Size, mobility, suppuration.

XI. **Urine**—Frequency of micturition. Quantity, colour, reaction, specific gravity. Albumen. Sugar. Deposit. Microscopical and chemical characters of deposit.

XII. **Generative System**—Menstruation, frequency, duration, quantity in excess or otherwise, whether painful, other discharges. Conditions of uterus and pelvic organs (when examination is possible).

XIII. **Treatment**—Prescriptions and diet to be entered, and all alterations noted with dates.

XIV. **Progress of Case** to be noted as occasions may require. Pulse should be recorded whenever the temperature is taken. Urine must be noted at least once a week.

When important symptoms or complications occur, besides a description in the text, an indication should be given in the margin. As a good general hint for note making, *remember in all cases, without exception*, a note should be made on the first day after admission; in all acute cases a note should be made every day while the symptoms persist; after this, notes may be made less frequently, but any **fresh** symptom or complication must be immediately noted.

XV. **Result**—A description of the patient at the time of discharge, or the mode of death, should be given at the termination of the case. This is, in some respects, the most important part of a Clerk's duty, and *must on no account* **ever** *be omitted in any case*. The description need only be of that system or systems which have been affected by the disease.

The Clinical Clerk to sign his name in full on completion of the case.

At the second step diagnosis frequently stops short; possibly, though let us hope rarely, from carelessness or indifference on the part of the observer, as when a cough or diarrhœa is treated by a dose of opium (reluctance on the part of the patient to submit himself to thorough examination has sometimes too to be overcome); more frequently and excusably because further information is useless in the present state of our therapeutical knowledge, or because some very obtrusive symptom must be immediately subdued

if the patient's life is to be saved even temporarily, as may happen in hyperpyrexia from any cause, or in the agony of calculous colic; most frequently and really unavoidably because the further information required is absolutely unattainable until death has afforded the opportunity for more thorough investigation, as in a case of severe hæmoptysis, for example, or rapidly fatal septic pneumonia or perforative peritonitis. Like the first step, this second one is in many cases a very simple matter, but to the student and young practitioners it is apt to appear easier than it really is. Nothing but wide experience by the bedside and, above all, in the post-mortem room, will convince us of the difficulties which surround even the simplest *accurate* diagnosis, and there can be no greater mistake than that of at once accepting the most obvious as the most correct.

Apart from the above exceptions and difficulties, it should always be our endeavour to take the third and final step in diagnosis. Of its importance generally in clinical medicine examples are almost superfluous, the point is so universally admitted. I need only mention the flood of light thrown on an obscure case of albuminuria by the recollection of a forgotten attack of — it may be mild — scarlet fever, what a relief to our anxieties it is to find a bad sore throat as a precedent fact in a case of squint, and, *per contra*, how the discovery of the bacillus tuberculosis in the sputum redoubles our anxieties about what was previously looked upon as a simple catarrh. But beyond all this the discoveries of the last quarter of a century in bacteriology have made this third step of much greater importance still, for they have held out to us hopes of cure in many cases hitherto incurable. Whether these hopes are destined in the future to be realised or disappointed it would be rash to prophesy, though certainly the results hitherto obtained are encouraging. As the basis on which these hopes are founded is very germane to the completion of a diagnosis, we may here enunciate its principal points. Stated in order the argumentative propositions run thus: —

(1) Many groups of symptoms or diseases are caused by microbes invading the body.

(2) In many of these, if not in all, the actual symptoms are caused by a toxin or toxins manufactured by the microbes, either directly from their own metabolism or indirectly from the tissues of the victim.

(3) The spontaneous cure of such symptoms is, at least in some cases (possibly or probably in all), effected by an antitoxin or neutraliser of the previous toxin; this in turn

may apparently be produced either by the microbes or the tissues.

(4) Such antitoxin can be obtained, with more or less success (by methods which need not here be entered upon), in quantities and condition available for therapeutic uses.

(5) It should follow, and has in many cases already been proved to follow, that the injection of such antitoxin, if effected early enough in the disease, is efficacious in curing the patient.

These propositions would appear to be scientifically demonstrated, but there is one point which is still in doubt, and that the most important one from our present point of view; it is this — does each separate and identifiable microbe produce a special and specific toxin, *i.e.* a poison of definite composition — as definite, say, as morphine or other alkaloid — peculiar to itself and requiring for its neutralisation an equally definite and specific antitoxin? or, on the other hand, do many different microbes produce a common toxin, so that the antitoxin produced by one microbe or method of preparation can be used for the toxin of another kind of microbe? All the evidence — at least the weight of evidence — we at present possess points in the direction of the former of these two suppositions being the true one. Witness the almost conclusive clinical experience in the treatment of diphtheria, of rabies, and of tetanus, as well as the brilliant successes and the disappointing failures in cases of septic disease, in which the particular microbe at work is less definite or less familiar, and where success has been compulsorily attributed to the right antitoxin, failure being due either to the wrong antitoxin or to a too late application. I think, therefore, we shall be obliged ultimately to admit its truth, and we can see at once on this admission how enormous is the importance of the third step in diagnosis, to be able to say, "This case of illness is due to this particular microbe, and I must use its antitoxin as quickly as possible."

Besides this therapeutical use of a completed diagnosis, the principle of toxins and antitoxins has lately been applied to that completion itself, *vide* Typhoid Fever. May we not hope in the future to see the two procedures more frequently working hand in hand, complementary to one another?

Causes of Disease in General

In the special sections of this work we shall always be considering diagnosis as an induction from symptoms. We may here in the

introductory or general chapter briefly review the matter from the other standpoint, and see what are the causes of disease and the general processes by which these causes produce the symptoms which we have later to analyse.

The fundamental causes of disease belong to but few really separate categories, which may be presented in comprehensive forms as follows:—

Causes of Disease

A. From without (environment in the larger sense):—

Coming from the world at large

- Traumatism
  - Chemical (on the surface of the body).
  - Grossly physical effects, and those of excess of heat and cold.
- Parasitic
  - Macroscopical or coarsely microscopic
    - External — fleas, lice, bugs, etc.
    - Internal—worms, etc.
  - Finely microscopic (microbes)
    - (1) Essentially pathogenic, or (2) rendered so by peculiar opportunities of vitality or food supply.
- Poisons
  - Recognised as such — arsenic, alkaloids, ptomaines, etc.
  - Ordinary articles of food rendered injurious by circumstances of too free indulgence, imperfect mastication, decomposition, etc., and also by unusual changes in digestion with absorption of poisonous ptomaines, etc.

Coming from the immediate surroundings of an organ (its local world)

- Perforation of a hollow tube from any cause whatever.
- Direct extension of processes of disease from one organ to another.
- Carried in the blood or lymph stream, convected from place to place.
- Forced malpositions, as twisting of tubes and dislocations of more solid organs.

B From within (idiopathic, so to speak):—

(*a*) The more or less direct effect of altered function in one organ upon another organ, *e.g.* the inter-

related functions of liver, stomach, kidney, pancreas, etc.

(*b*) The effects of wear and tear, age in its physiological sense.

(*c*) Congenital or inherent debility of tissue either absolute or relative to the work demanded of it.

(*d*) New growths of intrinsic formation.

That the apparently innumerable causes of disease should be reducible to such a small number of independent types is at first somewhat startling, but I think a little consideration and comment will show that the above errs on the side of excess rather than defect, and that some of the groups overlap.

Traumatism.—In its widest sense—of injury—this would include nearly all the other causes of ill health which arise from the environment of the individual. Food imperfectly masticated or otherwise by its *physical* qualities unsuitable for digestion is, for example, very likely to cause a wound of the stomach with its attendant evil consequences. In its grosser sense the cause belongs almost entirely to surgery. We may recognise its probable single action when health speedily returns after the removal of an exciting *causa causans*. Mr. Hutchinson would refer most skin diseases to this category or the next.

Parasites.—Of the grosser forms of external parasites the action is mildly irritative or traumatic, increased largely by scratching or rubbing. If through the wounds thus produced microbes find an entry to the blood or lymphatic vessels—the skin teams with ever watchful enemies of this character—the result may be as bad as, in fact is identical with, the other microparasitic troubles, the skin, instead of some mucous or other surface, being the point of entry which is thus more easily traceable. Of the larger internal parasites—collectively denominated worms—the results are more varied. Thus round and tape worms may reside within us without causing any trouble whatever, but they may cause much irritation with reflex symptoms, they may block passages, *e.g.* the common or cystic bile duct, or cause tumours (hydatids), or by migration of the young (*Trichina spiralis*) cause serious symptoms in any organ; the *Anchylostoma duodenale* may produce profound anæmia by abstraction of blood.

To the second—finely microscopic—group of parasites the ever-multiplying discoveries of medical science are rapidly relegating every disease to which human flesh is heir. Possibly enthusiasm

rather than hard facts is having something to say in this matter. When present in numbers, or with virulence sufficient to create a pathological disturbance in the body, they have one almost invariable and common result, viz. pyrexia (in primary syphilis, in gonorrhœa, and possibly a few others — chronic rheumatoid arthritis, for example — this is least marked, and even in tubercle it is doubtful how much of the fever is due to the specific bacillus), a result to be expected when we consider the enormously preponderating influence which metabolism has in the production of our supply of body heat, and then remember that in microbic disease either a toxin or a contagium vivum is, or may be, carried all over the body, stimulating every part of it to a metabolic antagonism.[1] This pyrexia itself, however, almost immediately offers points of difference aiding diagnosis, in suddenness of onset and rise, in duration, in irregular variability or regular periodicity; cf. typhoid and typhus, scarlet fever and measles, malaria and general septicæmia, etc. In many cases, too, the seat of inoculation or of first morbid appearances is available, the uterus in the puerperium, the tonsil in diphtheria, the genito-urinary apparatus in venereal diseases. Still later we have the selective seat in which the poison has left its traces, such as the peripheral nerves in diphtheria, the kidneys in scarlet fever, the spleen in malaria, etc. Further points will be found in the special sections.

Poisons. — Poisons, as ordinarily understood, may be divided into three classes: (1) Those which act upon the alimentary canal, and are practically not absorbed, *e.g.* all corrosives and many irritants; (2) those which only act after absorption, *e.g.* most alkaloids; (3) those which act in both ways typically, phosphorus, arsenic, and oxalic acid. The first and last classes will show alimentary symptoms in excess, vomiting and diarrhœa within a short time of being taken, and have one point in common, in marked contrast to the common symptom in microbic invasion: this is a subnormal temperature, largely induced by shock and mental anxiety. The second class in its entirety, and the third in the later phases of a case, show appropriate features according to the selective affinity of the poison for special organs, digitalis for the heart, conium and strychnine for the motor nerves, phosphorus for liver and muscles, opium and belladonna for the cerebral cortex, etc., and thus the case may be provisionally or even finally diagnosed in conjunction with the history.

[1] This metabolic activity or antagonism not only explains the phenomenon of pyrexia, but also lies very close to the root of what (*vide* Processes of Disease) we know as inflammation.

When articles of diet become injurious, they too, like the more virulent poisons, produce symptoms in one of two ways: (1) by acting as immediate irritants to the alimentary canal, with vomiting and purging, as in ptomaine poisoning, or in actual surfeit of food otherwise wholesome; (2) by pouring into the blood or lymph stream a greater supply of nutriment than can be adequately disposed of in the great chemical laboratories of the body; hence arise, more immediately, unpleasant sensations in the shape of sleepiness, biliousness, headache, etc., and remotely, a storing up of an undue amount of fat — obesity — flabbiness of body and mind, general lassitude, etc., and perhaps more remotely still, gout and goutiness, with all their pains and penalties, and possibly also a lessened resistance to attacks of microbic troubles.

Immediate Surroundings. — From the little world of its own special environment every internal organ, which has no immediate and direct communication with the larger external world, gets all its serious troubles. The blood and lymph that bathe its tissues bring with them the chemical or vital poisons that interfere with its physiology, or cause grosser mischief still, *e.g.* nephritis, acute yellow atrophy, meningitis, endocarditis, etc.; the perforation of a tube allows its contents to become a serious source of irritation to neighbouring structures — gangrenous inflammation from extravasation of urine, for instance, when ureter, bladder, or urethra is ruptured, or peritonitis when the contents of the alimentary canal are allowed to escape freely into the peritoneal cavity. Inflammation has but slight respect for the fascial or fibrous boundaries laid down for its guidance by anatomists, but spreads with but little hesitation from meninges to brain or cord, from endo- or pericardium to myocardium, from stomach to liver, etc. Displacements of the heart by pleural effusions or more solid growths will cause serious interference with the circulation; a displaced kidney brings about a great deal more than simple mental worry; twisting of the gut, or even of a pedicelled tumour, may, and often does, bring about a fatal illness. All these are illustrations of the working of the internal environment, and yet there are but few of them which with equal propriety might not be put under the heading primarily of poisons or traumatism.

Arising from within — Idiopathic Diseases. — It is only by courtesy — because we cannot always discover the original culprit — that disease in one organ, almost certainly arising from the wrong-doing of another, can be called idiopathic, or that this group can retain its position amongst the primary causes of disease. We often

speak of a primary congestion of the liver, or of an idiopathic anæmia, but in both cases it is extremely probable that the alimentary canal is the first offender by passing an excess of suitable, or some unsuitable, material to the liver or blood, which in turn exhibit the symptoms of complaint; and even now we have to throw the blame rather on the individual, who has by over-feeding or neglect of defæcation originally induced a plethora of nutrient material or absorption of poisonous products of food decomposition unavailable for nutritive purposes. In pneumonia and bronchitis danger but rarely arises from obvious asphyxia; it is almost constantly due to the difficulties the heart experiences in keeping up the circulation. This in turn may be due to the imperfect oxygenation of the blood by the lungs. Still the lung trouble is originally derived from without. When the functions of the kidney are imperfectly performed every organ in the body feels it, owing to imperfect removal of waste; yet the kidney itself has suffered for the fault of stomach, or from the introduction of microbic toxins. In fact, the more we inquire into this group of causes, the less the reason for retaining it, and the more does it divide itself up amongst the other groups.

Wear and Tear.—In wear and tear we get the first trace of disease really independent of external conditions; they are the essential conditions under which all living things exist. It is a common idea with the laity that in seven years every cell of the body is changed in its constituent molecules. As far as I know, there can be no exact foundation for this belief, but it probably, for all that, contains a pregnant approximation to the truth, and should loss—however gradual—be not equalled by repair and replacement, function must suffer, with the inevitable reaction known as dis-ease. The deficit in childhood is evidently nil, and as age advances, with waning elasticity in power of repair, it may be extremely minute, so that many years may elapse before the resultant loss of function is sufficient to attract attention. I think it probable that many cases of so-called cirrhotic kidney—and a better illustration of a chronic insidious disease could not be given—are thus induced. The kidney mischief—want of repair in epithelial cells—acts and reacts in turn upon the vascular system, where indeed, in the shape of arterio-capillary fibrosis and atheroma, the effects of age are more visible and pronounced, and also more serious than in any other organ or tissue. This vascular degeneration operates in two ways: (1) directly, the vessels themselves becoming brittle and absolute rupture taking place, or by losing

natural elasticity[1] they gradually yield to the blood pressure within them, and aneurysms, large or small, single or multiple, form, with all their attendant dangers; (2) indirectly; the minuter vessels by their pathological changes either (thrombosis) starve outright the tissues to which they are distributed, or (by thickening and alteration of wall) interfere with that due interchange of nutrient and waste material which is the life of the tissues, and thus lead to such impaired vitality that the tissues cannot adequately resist untoward influences — toxins, etc. — nor properly repair damage inflicted upon them. It is by such considerations as these that we are led easily to understand how in old people or those prematurely worn out (alcoholic excesses, physical toil, chronic lack of nourishing food) the prognosis of disease is made much worse, and also to appreciate why symptoms in such patients may be unexpectedly obscure or insidiously vague; pyrexia, that constant accompaniment of metabolic resistance and cell warfare, may be but slight, or even absent in cases which would otherwise be termed acute; the foundations of life may be sapped and death intervene while we are still waiting for the explosion of general and local manifestations that should tell us where the enemy is attacking; a smouldering pneumonia, an apyrexial empyema, a chronic and insidious uræmia will illustrate the point.

Congenital or Inherent Debility of Tissue.—Considered strictly, this whole group can only be a particular illustration of the influence of the immediate environment, of the physiological—or pathological rather—influence of one organ upon another, for during intrauterine life the fœtus is as much a part of the mother as her kidneys or heart, and is as much dependent for its health and integrity upon the due interrelation of her organs as are those organs themselves dependent upon one another. For more practical or clinical purposes, however, the matter assumes a different aspect, and we may divide these into three fairly distinct categories. First, those cases in which an active process of disease is going on in the child at the time of birth, a disease which has actually attacked it—been transmitted to it conveys a slightly different impression—while it was part of the mother. Syphilis and the acute specific fevers offer us typical examples of this, and recent reports would seem to render it possible, if not probable, that tubercle may in a similar way attack a child *in utero*. Simple anatomical deformities might

[1] It must be remembered that elasticity essentially consists in a "power to return *to an original form* after a distorting force of any kind—pull, pressure, or twist—has ceased to act."

be placed in the same class. Secondly, those cases in which inherent defect of tissue is assumed—rather than proved—to exist to explain certain so-called idiopathic affections. A child, for instance, is born healthy to all appearance, and remains so for years, and yet without any ascertainable cause disease makes its appearance in certain structures or organs; mental deficiencies, primary sclerosis of parts of the nervous system, primary myopathies, hæmophilia, would be examples of this class, amongst many others, in which we invoke heredity as a *deus ex machina*. Thirdly, the principle may with a little extension be used to explain those delightful words, "idiosyncrasy," "dyscrasia," "diathesis," etc., which are such beautifully simple ways of explaining (!!) our ignorance of etiology. Thus, a number of people take a similar dose of a drug; some exhibit marked effects, others none. Why? Oh, idiosyncrasy! Again, a dozen individuals are exposed to the same unusual disturbance of gross environment; one gets acute rheumatism, another nephritis, another pneumonia, another pleurisy, another chorea, and some show no trace of the storm. Why? Oh, different diatheses is the glib reply. However, explain or mystify the matter as we may, the facts remain, and we utilise them clinically when we insist upon the importance of careful inquiry into the family history of a patient, and into the previous illnesses that may have occurred of a similar nature to that for which our advice is sought. The layman puts the same idea into different words when he exclaims, "The old doctor is the man for me; he understands my constitution."

New Growths.—Of the exact etiological factors underlying the *starting of a primary* new growth, our real knowledge up to now amounts to — nil. Pathologists were lately — and perhaps even now are—keen in the pursuit of a hypothetical cancer microbe—a psorosperm, it was called. If such exists, its entrance, its life, and its death are still mysteries to us. The only clinical causes we can yet appreciate are blows as the starting-point of many sarcomata, and chronic irritation for many epitheliomata, but we still await anything like an explanation of the vast bulk of growths of a malignant — or innocent, for that matter — nature. Cohnheim's theory of fœtal remnants is perhaps as fascinating as any; but even this goes but a little way.

Processes of Disease

However few the really independent causes of disease are thus seen to be, the individual morbid processes which they excite are

still fewer, for they overlap one another even more than the causes, both in their intermediate phases and in their final results. Of intermediate phases only two are possible. Every organ and tissue in the body possesses two primary attributes—structure and function, —and the processes of disease can only exhibit themselves as interferences with one or the other, or more probably with both, of these attributes; the diagnosis and discrimination of these interferences constitute the whole of our morbid anatomy and physiology. With microscope and naked eye we attempt, after death, or in fragments removed from the living, to find and to study the alterations in structure, and with the clinical eye we endeavour to penetrate the mysteries of altered function. Our instruments of sight, natural and artificial, aided even by most elaborate staining processes, are very imperfect for studying the finer — molecular? — structural changes, and hence, though much has been revealed, very much more remains to be discovered. But the omissions are the less important from a clinician's point of view, because, firstly, we cannot be sure that what is seen was there during life; and, secondly, because of the limited capability of nature and the still more restricted power of drugs to restore structural damage, even when it can be detected or surmised. We cannot, moreover, determine what is reparable and what is not, except by that means which is our only therapeutical hope, viz. by endeavouring to promote to the utmost harmonious function, the loss of which is indeed but too frequently our only means of guessing that structure has suffered. The power, on the other hand, of the clinical eye is only limited by the amount and character of the intelligence which can, by reading and experience, be brought to bear upon unravelling the intricate web and woof of symptoms, upon determining which of them are primary and essential, and which are secondary or of less moment; which we can directly influence, and which must be left to the control of other organs.

The gross or comprehensive processes by which these interferences with structure and function are brought about are:—

1. Inflammation.
2. Degeneration.
3. New growths.

Inflammation.—Into the microscopic details of diminished drainage from, and excessive entrance of blood and serum into, an inflamed area or organ I do not propose here to enter, nor into the diapedesis of white and red corpuscles, nor to the differ-

ence between these cells and pus, nor into the vexed question of the origin of fibroblasts. Collectively, all these constitute the structural changes which we can see, and the student is too apt to lose himself in these details of a microscopic slide and not to think of the *functio læsa* of vessels and tissue cells which, from the medical, as opposed to the surgical, point of view, is the chief, if not the only, object that requires attention. It is true that we frequently call upon a surgical colleague, and rightly too, to empty by artificial means a serous cavity which has become filled with the products of inflammation, but it is immaterial to a clinician whether the nucleated cells that appear at a focus of inflammation are professional policemen or special constables sworn in for the occasion; it is immaterial whether repair be executed by builders called in for the purpose from a distance, or by the inhabitants living in the neighbourhood; they all have to be nourished by the food in the blood, they all are worried by similar irritants, and our only concern is to remove the irritant as speedily as possible and do our best to keep the blood in good condition.

The *functio læsa* of capillary blood-vessels leads to excessive, insufficient, or improper food supply to, and imperfect drainage from, the special or specific cells of a part; this leads to altered vitality in these cells, and this in turn is exhibited by disturbance in their function—excess, perversion, or diminution,—and eventually, if the trouble be severe enough or long enough continued, the cells die outright. It is thus that we must think of inflammation in its earlier active phase—kidney cells treated in this manner allow wrong materials, albumen, etc., to escape in the urine; they fail to pass on the right substances, or even cease to secrete at all; hepatic cells cease their glycogenic and bile formation functions, or perform them wrongly; the cells of the alimentary mucous membrane excrete vitiated digestive fluids, and when digestion has thus been improperly performed, absorption becomes a harmful process, the gut muscle too is hurried into excessive action, and diarrhœa ensues, or, if it be involved seriously in the trouble, its function ceases, and paralytic obstruction—the most serious of all forms of recoverable obstruction—occurs; in the lungs excessive secretion or effusion fills the air cells, and thus brings about cessation of the aerating function; imperfect nutrition of nerves sends them astray in functioning, and their property of irritability becomes excessive, insufficient, or perverted, and so the illustrations might proceed through the body.

The *functio læsa* of the tissue cells may thus be secondary to

that of the capillaries, but more frequently in medical practice we see it as a primary phenomenon due to a toxic condition of the nourishing plasma which surrounds them; under these circumstances the cells cease to a large extent their own specific activities and take up ones that are foreign to them, manufactory of an antitoxin and other unusual metabolic processes. When thus struggling against unnatural conditions they are very liable to be attacked by fatal structural changes unless relief be speedily afforded them. It is thus I understand the evil effects of the working of blood-poisoning, whether this be generalised over the whole body, as in zymotics, or whether it arise from some quite localised process, as in a poisoned wound with absorption therefrom.

When these destructive phases of inflammation have ceased and nature exhibits her limited power of parenchymatous-cell reproduction, we must remember that the new or young cells have to learn their business and get accustomed to variations in their environment; they are likely at first to be of feeble vitality and resisting power, and chronic exfoliation may thus be kept up till irreparable damage is done to the organ of which they form the essential constituent.

And lastly, when repair is complete we have to realise the strangling effects of the new-formed fibrous tissue, which, in us, is the best that nature can do to fill up a gap. This fibrous tissue cannot pick out urea nor biliary constituents from the blood, it cannot absorb peptones or fats or oxygen, it cannot transmit messages, nor can it contract and relax in response to messages received; it has only one function, that of contraction into a condition of stable equilibrium, and this it exercises with most pernicious effects on vessels and parenchymatous cells, strangling the one and starving the other.

I have written at some little length on the local processes of inflammation in the hope of making it clear that a scar in the kidney is identical with one in the lung or liver, that a catarrh of the bronchi is identical in its processes with one of the gut; that, in fact, the principles once grasped, details can be evolved with readiness for special cases, and pathology thus changed from a heterogeneous collection of wearying facts to a splendid illustration of fundamental laws working on lines variable within small and special limits.

Degeneration.—In text-books of pathology it is usual to describe this process as either a primary or a secondary phenomenon, and then to give particulars of many varieties: amyloid, brown,

glassy, cloudy, fatty, mucoid, colloid, fibroid, etc. Important as this knowledge is from many points of view, it may for our present purposes be largely ignored, for both forms and all varieties mean clinically very much the same thing, viz. diminution in vitality and functional capacity, and ultimately death, of the parenchymatous cells, whose fate is being discussed.

The primary form, strictly speaking, has for its underlying essence either natural wear and tear, or congenital and inherent poor quality of material, both of which have been already alluded to as primary causes of disease; but it is usual in clinical medicine to think of and accept as primary many varieties of degeneration, fatty, etc., which are in reality induced by an insidious form of chronic poisoning or starvation through the blood supply, which in its turn is influenced primarily by the alimentary canal and the substances put into it and absorbed thence. These are thought of as primary because it is difficult, and perhaps impossible, in some cases to appreciate the alimentary errors which are at the bottom of the matter. In other cases of so-called primary degeneration typically in the nervous system the death of the (neuron) cell, and the consequent atrophy and degeneration of its processes, may be brought about by the agency of very insidious microbic toxins, syphilitic for example. This probably is the explanation of many cases of tabes dorsalis, and other primary affections of the nervous system not directly traceable to inherent weakness.

To see with the actual or with the clinical eye the process of secondary degeneration is usually comparatively easy and simple; for whereas in the primary form the trouble arose from insidious qualitative changes in the supply of nutriment and the removal of waste, here it is due either to a gross interference with the quantitative nutritive supply, or to an equally gross mechanical interference with the protoplasm of the cells—squeezing or distortion. A few illustrations will make the position clearer. The absolute blocking of a large (or microscopical) artery by thrombos is, embolus, or ligature, leading to coarse (or fine) gangrene or necrosis of tissue, is the most obvious illustration of the former process. It is well seen and very common in the brain, spleen, kidney, or lung. It occurs, though less commonly, in the intestine, liver, and other organs (in surgery it is common enough in limbs, or part of them). The secondary degeneration of the processes, long and short, of the neuron cells of the nervous system is a less obvious but equally certain illustration; it differs in that the nutritive supply or stimulus reaches the process (and indeed probably also gland cells, muscles,

etc.) through other channels than obvious vessels, but the degenerative result follows as surely as does general wasting of the body at large when the stomach is seriously incapacitated. The actual rupture of a vessel may act in both ways; it is certain that the blood supply to the part concerned is stopped, and at the same time the blood, escaping from the vessel often at high pressure, mechanically tears asunder the cells of the tissue and exposes them to every form of distortion and violence. Inflammation, again, in its reparative stages, is a potent cause of secondary degeneration through the cell strangulation its scar contraction brings about (in its acute destructive phases the destruction is wrought by the action of poisons and excessive reaction to them), and this not only in gross cases of localised trouble, but also in the more chronic and diffused forms, as, for example, in cirrhosis of the liver of alcoholic origin, in pneumonokoniosis and other forms of cirrhosis of lung. The influence of cardiac disease (*q.v.*), with its back-pressure stagnation and increased transudation from the capillaries, acts in a very similar manner to the diffuse forms of inflammation—loss of function, then death, with a very imperfect or altogether absent power of reproduction of cells of highly specialised function, this in turn followed by a fine fibrosis to fill the spaces left by the absorption of dead cells—these are the processes induced in organs and tissues by cardiac failure.

New Growths.—But one diagnostic difficulty here presents itself, and that is of establishing by physical signs the presence of a malignant growth. This is frequently great, and sometimes even insuperable without surgical aid (will the Röntgen rays ultimately assist us here?); but whether this difficulty be overcome or not, the pathological processes which a malignant growth starts in the body are comparatively simple to understand. Thus a malignant growth acts:—

Mechanically or biologically	}	coarsely or insidiously.

Of the coarse mechanical form of trouble the blockage of a hollow tube, either by projection into its lumen or by pressure from without, is at once the simplest and most common illustration. Thus the intestine, ureter, or bile-duct may be blocked, and gastric distension, with vomiting and emaciation, or intestinal obstruction, with retention and absorption of fæcal products, occur, or retention of urine or bile, with a more or less rapidly following toxæmia by

products that should be discharged from the body. Arteries, capillaries, veins, and lymphatics suffer in precisely the same way, with resulting anæmia, or even gangrene, œdema, or effusion, simple or chylous, according to the function of the blocked vessel. Solid organs, too, may suffer from the same gross pressure, with disturbance of function and distortion of structure. The brain gives us a common illustration of this class, but, owing to the fact that it, with its vessels of all kinds, are enclosed in an unyielding box, the results are complicated, for they may be due to direct pressure on specialised cells, as well as to indirect effects of altered circulation.

More insidious, but equally real, mechanical (pressure, strangulation, disturbed circulation) effects are produced by the creeping processes of the growth, and explain the symptoms and cause of death in many cases, thus in the lung and liver, structure and function frequently suffer far out of proportion to the *naked-eye* bulk of the tumour. In the intestine the muscle is paralysed by these processes, and so contributes by this paralysis a large share to the obstruction. In the brain, too, it is thus that we must explain the fatal effects of a small tumour growing into a very important area of the organ. This insinuation of the growth into a tissue, with violent separation of the elements, frequently sets up a diffuse inflammatory reaction in the cells of an organ, and this must also be reckoned amongst the insidious mechanical effects.

The biological or vital morbid processes started by and in tumours would seem to depend somewhat upon what we may call the physiology of new growths, which may be thus summarised:—

1. They possess a great power of increase in bulk, or vitality of reproduction.

2. They would seem to possess some form of internal secretion or metabolic activity whereby chemical products are poured into the blood stream — products which are foreign to the normal tissues, and apparently of considerable power for evil in some cases.

3. They appear to possess but little or even no power of resistance against irritative, especially microbic, influences.

(1) has already been considered as starting all the mechanical effects, both gross and fine, of tumours. It also explains (microbiological researches may alter in some ways this view) the phenomena of recurrence after *apparent* extirpation, and metastases due to the transference from place to place of cells endowed with this special power of reproduction.

(2) would appear in some measure to explain the anæmia and cachexia which frequently, but by no means invariably, accompany

the existence of a malignant growth. Starvation and the mechanical effects mentioned above doubtless play the chief rôle, but many cases occur in which these are not sufficient to explain the great cachexia occasionally met with even when the growth does not affect an important organ. Thus does a growth insidiously undermine health.

(3) It is to the feebleness of the powers of resistance (the protective reaction to irritation possessed by normal tissues) must be largely attributed the ease with which malignant growths become the seat of destructive phases of inflammation, with but little corresponding power of allowing or promoting the reparative phases. When microbes invade, as they so frequently do, malignant neoplasms, they are but seldom killed by the growth, or even diminished in vitality, and thus it very frequently, indeed, happens that secondary septic processes carry off a patient who begins by suffering from carcinoma; thus in epithelioma of the lips or tongue death is very often due to inspiration-pneumonia; and, indeed, in carcinoma of the lung it is as often as not a pneumonia that finishes the scene. In carcinoma of the gut, peritonitis is to be feared long before perforation can occur, as though the growth encouraged the penetration of microbes or their toxins. Such exhibitions may with justice be termed the grosser forms of biological active processes induced by growths, an empyema or a purulent peritonitis being the immediate diagnosis.

We have thus completed an outline sketch of the primary causes and processes of disease. From these, physiological deduction will lead us to expect certain morbid phenomena as the result of certain untoward influences in our environment. We must now proceed to discuss diagnosis by inductive methods, accepting the morbid phenomena (the physical signs and symptoms discovered in or complained of by our patients) as the threads of Penelope to lead us out of the maze, or to discover the fountain-head, and thus apply therapeutical measures with some idea of what results we hope or intend to produce.

CHAPTER II

NOTES ON A FEW OF THE TERMS USED IN MEDICINE AND MEDICAL DIAGNOSIS

Physical Signs *v.* Symptoms

Symptoms are those uneasy or abnormal sensations or feelings of which the patient complains either spontaneously or as the result of verbal inquiry, *e.g.* pain, cough, shortness of breath on exertion, etc. It is customary also to include many other phenomena in the term, such as the results obtained by chemical, bacteriological, or microscopical research, and even some unusual conditions of the nervous system assuredly only found by physical examination, *e.g.* absent knee jerks, etc., but I think it well to have a sharp line of distinction where possible.

Physical signs is a term used to describe those phenomena of disease (especially of the chest and abdomen) which only become apparent to the observer's senses by means of what is termed a physical examination, including, in its completest extent, inspection, palpation, percussion, and auscultation in their simple and modified (*e.g.* succussion) forms.

If these definitions are not in every particular absolutely satisfactory, they at least form a sound and workable conception for clinical purposes. That some such definition as the above is useful, my own experience would tend to show, for I have been asked the distinction between the two sets of phenomena in a court of law, and it is no unfrequent occurrence for a student to glibly enumerate a string of the easier and less important physical signs when asked for the more perplexing and intricate, and withal more germane, symptoms, a reply which may not unfairly be allowed to annoy an examiner and create in his mind a bad impression of a candidate's

clinical knowledge. Nevertheless, it must be borne in mind that neither in science nor in ordinary usage is there a fundamental distinction based on clear conceptions of existing facts. For example, pain may certainly be claimed as a symptom, and cardiac bruits as physical signs, but what shall we say of the pain-drawn face, or the cardiac bruit that is audible and troublesome by its noise to the patient? It follows clearly that where no pressure from counsel or examiner is brought to bear, the two terms will be used almost indiscriminately.

Pathology

No term in medical science is used in such a loose sense, and with so many confusing ideas attached to it, as this, and one cannot wonder at the student's difficulties in trying to answer a question such as, "Give, describe, or state what you know of the pathology of such and such a disease." "A chat about the etiology" seems at first sight to be a very happy definition, but frequently the term is used much more or much less comprehensively.

It cannot be too strongly insisted upon that there are at least three separate and, in some respects, different and distinct ideas contained in the word as commonly employed: —

1. The changes in organs, tissues, and fluids of the body to be found by the naked eye and microscope after death = morbid anatomy.
2. The explanation of symptoms (and often, indeed, of physical signs also) by reference to the known morbid anatomy; this = morbid physiology or the etiology (from within) of symptoms = symptomatology.
3. The explanation of the morbid anatomy *or* morbid physiology by reference to the action or influence of the environment on the organism = etiology (from without) of disease.

That all these three meanings may simultaneously be read into the word is obvious from its derivation (πάθος and λόγος), the theory of, or a discourse on, suffering; but its everyday, and especially its examinational, meanings are ill defined. For instance, the pathology of inflammation chiefly concerns itself with the naked eye, and especially the microscopical anatomy of the inflamed area and the changes going on in its neighbourhood, together with speculations as to the precise origin of the cells seen. The pathology of gout, diabetes, and blood dyscrasias in general, principally requires

a discussion of the primary seat of perverted metabolism, with a hypothetical explanation of the morbid physiological phenomena which arise in consequence of this perversion, morbid anatomical facts receding in the background as results rather than causes; while, again, the pathology of pneumonia or other specific fever would be very incomplete without considerable reference to the micro-organisms which cause them, and the modes by which these parasites enter the body and attack the tissues.

If no suggestions are given as to the precise form of answer required, a clear exposition of the pathology of any disease is best given by a brief, concise, but complete as possible, summary of the main (anatomical, symptomatic, and etiological) *facts* observed in its history, followed by a statement or discussion of those *theories* which have been propounded as best explaining or linking together these facts.

As an example we may sketch an answer to the question, "What is the pathology of cirrhotic kidney?"

The most important *facts* observed in connection with cirrhotic kidney are :—

A. *Anatomical.*—The almost universal discovery on autopsy that hypertrophy of the heart and degeneration of arteries have coexisted with the cirrhotic kidneys.

B. *Symptomatic.*—The extreme frequency with which patients thus afflicted suffer, and even die, from uræmia, cerebral hæmorrhage, or intercurrent inflammation of some organ. The condition of the urine.

C. *Etiological.*—That such patients are almost invariably at or past middle age, or have been gouty, or suffered from plumbism, or suffered from repeated attacks of acute or subacute nephritis.

The theories suggested to account for the anatomy are: (1) Johnson's stopcock theory. (2) Gull and Sutton's arterio-capillary fibrosis. "Renal inadequacy" (a term associated with the late Sir Andrew Clark), especially if of long duration, will largely account for the symptoms, while the condition of the kidneys, as revealed by the microscope, give a sufficient explanation of this renal inadequacy.

Or, to take a shorter example, the pathology of Addison's disease :—

The only constant anatomical fact is destruction of the supra-

renal organs, and that almost constantly of a caseous, probably tubercular, nature.

Pigmentation of the skin is not nearly so constant.

Symptomatic facts are few and summed up in progressive debility and frequent gastric crises and a very poor pulse.

Etiological facts none constant, but tubercle bacilli often found in the glands.

A theory has then to be stated and argued out as to the functions of the suprarenals, interference with which can produce these symptoms; it must be compatible, too, with the fact that destruction of the organs, other than tuberculo-caseous, frequently enough does not produce the same symptoms.

Contagion *v.* Infection

These terms are often used to indicate different methods by which a disease is communicated from person to person. Unfortunately (as is so often the case with terms originally possessing one specific meaning), habit and common usage, but especially increased recognition of facts, have so broken down the limits between the meanings, that the words now only remain as puzzles for students. Contagion should mean transference by actual contact of person with person, while infection should be a more comprehensive term, indicating that the disease to which the adjective "infectious" is applied can be communicated from one individual to another, and should include contact as one method of such communication. The essential fact of clinical importance is the communicability of the disease; observation and laboratory work must teach us the "*how*," leaving the clinician to use such expressions as "communicated by contact," or "by microbes floating in the air and gaining entrance to a second individual either by means of milk, water, food, or by the breath, etc." Such expressions, if cumbersome, at any rate convey a definite meaning; and until science supplies us with more accurate knowledge, "communicable" is quite sufficiently understood to include all such terms as contagious, infectious, etc.

CHAPTER III

MICRO-ORGANISMS AND ZYMOTIC DISEASES

FROM communicable diseases to microbes is nowadays hardly a step in thought, for the researches of the last quarter of a century have taught us that the two are essentially effect and cause. In this work, dealing mainly with clinical problems and practical medicine, I do not propose to give more than the very briefest outline of those fundamental principles of bacteriology which have now become essential for all medical practitioners. Beyond those principles and their correct understanding I do not hold that it is necessary, or even desirable, that students or medical men in practice should go; the extraordinary intricate details and scrupulous exactitude required for the scientific laboratory working of bacteriology are too engrossing for, and demand too much time from, those who do not intend to devote their life to purely scientific work of this description. The healers of the sick must patiently wait for the results of laboratory work, recognising and using those which are immediately available for practice. These already represent a greater amount of accurate and minute knowledge of the causes and processes of disease than all the previous centuries have yielded.

Meanwhile the following numbered paragraphs contain the principles of the subject in a form easily assimilable, and only to be acquired from monographs by a very tedious process of analysis.

1. Micro-organisms do exist in countless myriads everywhere in nature where the conditions of moisture, temperature, and access of air are suitable.

2. Their genera and species are probably more numerous than those of all animals or plants visible to the naked eye.

3. They are too small to show generic or specific distinctions

in their organs, consequently the classification of them for scientific purposes rests on :—

(*a*) Gross outward shape—cocci, bacteria, bacilli, spirillum, etc.
(*b*) Their methods of aggregation and segregation.
(*c*) Motility or the reverse.
(*d*) Their chemical and vital reactions to and on their artificial environment, *e.g.* ærobic or anærobic, whether they liquefy gelatine with or without the production of gas, or whether they do not liquefy it, what nutrient medium do they best flourish in or on; colour and appearance of the growth, etc., etc.
(*e*) Their reactions to various staining and decolourising reagents.
(*f*) And most importantly, their influence upon their host after inoculation into a vein or tissues or serous cavity, etc., *e.g.* whether they produce any symptoms of ill-health or not, the character of the symptoms, and the anatomical results on autopsy of the animal, etc. In this way they have been divided into two broad groups, the pathogenic and the non-pathogenic, a distinction which frequently breaks down when microbes gain access to a part of the body in which they are not usually found, *e.g.* the bacterium coli commune is a natural inhabitant of the alimentary canal, but it *seems* to cause very serious mischief when it gets into the tissues generally.

4. Those now known to produce, and generally accepted as producing, disease (the pathogenic group), constitute probably only a minute fraction of the whole number of species.

5. To prove that one particular microbe and no other is the cause of a given disease or symptom-complex, the following conditions must be rigorously fulfilled :—

(*a*) It must be constantly found in the blood, tissues, or discharges of the individuals suffering from the disease in question.
(*b*) From this source a culture must be prepared in a sterile medium, and the particular microbe isolated and grown in pure culture for some generations.
(*c*) From such absolutely pure culture it must be introduced into a fresh *healthy* individual of the *same species* as the original sufferer.

(*d*) This inoculation must reproduce the disease in the second individual.

(*e*) The special microbe must again be found in the second animal in the same situations as before.

In the strict fulfilment of these laws are contained all the difficulties of scientific bacteriology. They can only be appreciated by those of experience, and to publish the crude and inaccurate results of the dabblers in the science is merely to waste paper, and to retard the progress of medicine.

6. Applied to man and practical medicine there is only one microbe which has gone completely through the trial and come out triumphant, that is the organism which produces cutaneous erysipelas. The reason why no others have been intentionally put to the test is self-obvious; but laboratory accidents (only too frequently), experiments on animals, and justifiable deduction have so far proved the position as to leave no reasonable doubt that a microbe is at the bottom of the following well-known diseases:—

GROUP I

Tuberculosis (of every form and organ). Pneumonia (? one only). Anthrax. Typhoid. Asiatic cholera. Diphtheria. Malaria. Leprosy. Gonorrhœa (? one only). Rabies. Tetanus. Plague.	Of which it may be said that the type of the disease is fairly constant, and that one special microbe has been identified and universally accepted as the *essential cause* of the malady.

GROUP II

Cerebro-spinal meningitis. Dysentery. Influenza. Measles. Scarlet fever. Small-pox. Typhus. Varicella. Whooping cough. Syphilis.	Of which we may say that the conditions and circumstances under which they occur, together with the regularity of their symptoms, leave no reasonable doubt about each being caused by a definite species or genus of microbe, though such has not yet been satisfactorily isolated so as to be *universally* accepted.

Group III

Pyæmia. } The diseases hitherto enumerated under lists I and
Septicæmia. } II have a group of symptoms and clinical courses which are so far constant and distinctive (rash, periodicity, anatomical products, etc.) as to deserve a specific and constant name, and to lead to the belief that one, and one only, species of microbe is the *causa causans*. In these two diseases, on the other hand, the symptoms (fairly constant, but with no absolutely pathognomonic feature), the course, and careful bacteriological investigation, all tend to raise a conviction that more than one, if not indeed several, species of microbes may be concerned. To satisfactorily and simply account for the manufacture of the words it is necessary to state briefly the clinical classification of *pathogenic* micro-organisms.

For this purpose, then, we may divide them into three classes :—

(*a*) Those which, under any circumstances of the patient, are able after entry to the body to grow into, attack, and destroy healthy tissues, and hence can be, and are, easily carried by the blood-stream to distant parts of the body.

(*b*) Those which, under no known circumstances of the patient, are able thus to attack healthy tissues, but cause symptoms by living and growing on dead or dying tissues, and from there discharging into the blood-stream doses of a chemical poison extremely active, indeed, but incapable of multiplication.

(*c*) Those which seem to be of an intermediate character, and depend for their activity and virulence to a large extent upon the conditions of vitality of the tissues upon which they alight, and also on their own temporary condition of vigour.

Pyæmia, then, is a disease produced by a member of class (*a*), and in strictness must include anthrax, and nearly all the other specific diseases.

Septicæmia is a disease produced by a member of class (*b*).

Artificial cultivations and experimental inoculations would seem to show that between all three groups no line of strict demarcation can be drawn ; but this does not entirely destroy the clinical value of the distinction in cases of disease. For when in a natural state the distinction can be moderately well made, and it is probable that clinical (as opposed to laboratory experimental) diseases arise from the invasion of microbes of a natural (to them) healthy activity.

7. Pathogenic microbes would seem to be able to attack tissues

in one or both of two primary ways with a subdivision of the second method.

(*a*) By a sort of hand-to-hand fight, microbe *v.* cell = phagocytosis.

(*b*) By a weapon in the shape of a secretion = chemiotaxis, about which weapon two theories exist:—

(1) The secretion is itself a poisonous chemical substance = a toxin.
(2) The secretion is of the nature of a ferment, comparable to trypsin, say, and = a toxinogen.

On the other hand the cell defends itself either—

(1) By bodily eating the microbe.
(2) By secreting a substance neutralising the toxin, or ferment, or the products of the ferment; in either case it is called an antitoxin.

This is the most natural place in which to insert the clinical reasons for suspecting that a disease is due to microbial influences.

(*a*) The occurrence of an epidemic of the disease in question, in which the logic of events seems to prove, or, at least, strongly suggest, that on the one hand all the cases arise from a common (aerial, aquatic, or telluric) source, from which all gross metallic, *e.g.* lead, copper, arsenic, mercury, or organic, *e.g.* decomposing food, poisons can be and are excluded; or on the other hand that consecutive cases arise from previous ones, the latter a mode of spreading often better noticed in the smaller sporadic outbreaks, where observation is closer and the methods of "catching" the complaint more easily distinguished.

(*b*) The existence of localities where endemic diseases flourish, where a purely local cause must be at work, but where, again, all metallic and ordinary organic chemical poisons can be excluded.

(*c*) In both these cases the *a priori* argument will be enormously strengthened by *a posteriori* reasoning when the common, local, or personal factor has been removed, and the disease has ceased to attack fresh individuals, and the removed factor has been shown to contain no organic or metallic poison.

(*d*) The character of the disease itself an active acute onset, followed by definite fastigium, with crisis or defervescence, and a complete restoration of the patient to health.

(*e*) The finding of a definite microbe in the blood, tissues,

fluids, or excretions of the patient. Modern discoveries have made this an almost necessary part of routine clinical study, *but the microbe must be put through the above strict scientific proof before its causative rôle will be accepted.*

Into the extraordinarily fascinating subject of attenuation of microbes, causes of immunity, and protective and curative inoculations I do not propose, further than the observations recorded on p. 31, etc., to enter. The work that has been done in this direction would fill many libraries were it all published, but it is entirely founded on the above principles.

We will now proceed to consider the differential diagnosis of the ordinary zymotic diseases of England.

For the student to attempt to learn by rote the apparently chaotic variations in the incubation periods, symptoms, complications, and sequelæ of these diseases seems to me a useless, difficult, and never-ending task, but to give a working hypothesis as to the *essential causes* of these discrepancies and variations is a comparatively simple matter, bringing rule and order out of confusion. I say hypothesis advisedly, because though we have every reason to believe that incidents occur in the body in precisely the same potential sequence as in the test tube, we have no absolute proof that they do so. In the test tube there is no variation from hour to hour in the bulk and quality of the cultivation menstruum, except such as are brought about by the mixture of bacterial products; there are no policemen leucocytes exercising their unceasing vigilance in getting rid of foreign substances; it is doubtful how far these cells and the renewal of the menstruum can fundamentally change the phenomena, apart from their undoubted power to hinder, delay, or prevent them altogether.

So far, then, as science has allowed us to follow the workings of microbes, we are scarcely using the language of metaphor or analogy when we speak of the invasion of the body by specific germs in precisely the same terms as we use when describing the invasion of a geographical country by a hostile nation; the differences between the engaging forces are almost nothing but those of size and the nature of the weapons used.

The first event, then, is an attempted landing of the invading forces. To say that the expedition may be wrecked by storms on sea or land is nothing more than to say that multitudes of malignant and pathogenic microbes enter the apertures of the body or alight on the skin, but are immediately swept away by the forces of respiration (sneezing, coughing, etc.), by food and drink, by smoking, by

accidental rubbing of clothes, by washing, etc.; that, in fact, they never get a foothold in the tissues or blood.

Date of infection. A successful landing is the moment of infection, and from now onwards we have the varying incidents of a campaign.

Incubation period. The invaders have to strengthen their position, increase their numbers (for our story it is immaterial that they do this with inconceivable rapidity by reproduction), and provide food; the time thus occupied represents the incubation period.

Reason why in an epidemic all exposed do not sicken; meaning of immunity; abortive cases also. Even now, or at a somewhat later stage, when the march through the country has begun (abortive cases), a complete miscarriage of the whole expedition may occur through (*a*) a want of suitable food in the district, or (*b*) the energy of the local inhabitants and first line of home defences, who may annihilate the invaders as soon as they emerge from their local camp. It is possible in either or both of these ways to explain immunity, natural or acquired, permanent or temporary only.

Malaise of incubation period. The slight excitement caused by the rumours of an invasion represent the malaise and slight symptoms of the incubation period.

Onset sudden or gradual. The relative strengths of the contending parties explain the nature of the onset. In one set the invaders are so powerful, or have become so numerous, that their attack and onward march cause the utmost consternation throughout the land; in the other they are so insignificant in strength or numbers that only a slight disturbance is caused at first, though later their power may be enormously increased by reinforcements, etc.

Ingravescence of disease and fastigium. As the invaders march through the country the war rages with increasing violence, until either, in repeated conflicts, or in one great pitched battle, the invaders are destroyed, or *per contra*, the defenders are completely defeated.

Defervescence, or crisis, or death.

Convalescence and death from exhaustion in it. When the invaders have been thoroughly routed comes the time for rebuilding cities, towns, and villages, and for recuperation of the inhabitants, and it may happen that the whole

	nation is so exhausted that this is impossible, and life ceases in the country.
Persistent debility, or even stronger than ever.	Rebuilding and renovation may remain for ever incomplete, or may be such as to make the country stronger than before.
Complications and sequelæ.	In such a war of invasion there may be found three well-marked classes of events :—
(1) Specific to the disease, *e.g.* otorrhœa.	(1) The attacks on the principal cities by the original invaders.
(2) A second specific disease succeeding the first.	(2) A separate descent upon the country of a different invading force, which, eating different food, or fighting with different weapons, may go through all the vicissitudes of the first force.
(3) Non - specific complications, *e.g.* abscesses from pyogenes, coli commune, etc.	(3) Turbulence and riot on the part of a malcontent native or alien population which had previously, under pressure from local authority, lived more or less quiet, and even useful, lives.

Dropping now the possible suspicion of using metaphorical language, we know for a certainty we have the following variables in every equation of infectious disease :—

1. The inherent constitution of the individual's tissues, cells and body fluids, including the presence or absence of certain materials for the growth of microbes, the power of manufacturing antitoxin, or of eating up these microbes.

2. The quasi-accidental temporary powers of the same at the time of infection.

3 and 4. Similar powers on the part of the invading microbes.

5. The actual number of the latter gaining admission (antagonism between different microbes landing at the same time).

Scientific laboratory work has proved beyond all doubt that each of these is of the utmost degree of importance, but it has not given us even an inkling as to the laws which govern the variations on the part of the body forces, and is only on the threshold of investigations into those regulating the microbes ; the strongest men often enough fall victims to epidemics while the weaklings escape, some in contact with the patients are smothered with microbes and escape, while for others or even the same persons at another time, a single contact is sufficient to produce a most virulent attack.

Although these unknown variables and their unknown laws make

any tables of comparatively little scientific value, the following may be found of some practical utility in clinical work as well as for examination purposes.

TABLE OF INCUBATION PERIODS

Anthrax	Have been known to develop within 24 hours of infection, though the annexed periods in days are very much commoner.	2 or 3 days.
Cholera		up to 15 „
Diphtheria		2 to 4 „
Influenza		3 or 4 „
Pneumonia		
Relapsing fever		4 to 10 „
Scarlet fever		2 to 5 „
Septicæmia		
Tetanus		up to 21 „
Yellow fever		up to 18 „

The following, arranged roughly in order of duration, are:—

Gonorrhœa	2 to 3 days.	Pertussis	6 to 12 days.
Typhus	2 to 12 „	Measles	8 to 12 „
Glanders	3 to 18 „	Malta fever	about 10 „
Typhoid	4 to 20 „	Rötheln	10 to 21 „

Variola	11 or 12 days (curiously constant).
Varicella	10 to 14 „
Erysipelas	10 to 14 „
Mumps	12 to 21 „
Syphilis	18 to 28 „
Rabies	40 days and upwards
Tubercle	Unknown, probably some weeks.

Our previous considerations have led us *a priori* to expect what we actually find, viz. that the variations are much too great and too irregular to serve any diagnostic purpose; but they explain why three weeks has been adopted as the period of segregation of individuals who have been exposed to infection, where such segregation is possible, as in schools and public institutions.

When the incubation period is at an end, and the actual onset of the disease begins, diagnosis rapidly narrows itself down. Many of those enumerated above show characteristic features, apart from pyrexia, almost at once, thus:—

Anthrax	Ring of vesicles with central black slough.
Cholera	Characteristic diarrhœa, except in fulminating cases.

Diphtheria	Membrane on infected spot.
Pneumonia	Cough and disturbance of respiratory rhythm, *vide* p. 35.
Wound-Septicæmia	Wound inflamed and discharging.
Gonorrhœa	Urethral trouble.
Syphilis and venereal poisons	A chancre of some description.
Glanders	Particularly acrid nasal discharge.
Erysipelas	Sharply defined red blush surrounding a wound.
Tetanus	Stiffness of jaws, or abdominal muscles.
Mumps	The swelling of both parotids.

The bulk of the remainder is constituted by the common so-called zymotic diseases or exanthems of our country. In them the presence of an epidemic gives the primary and the strongest clue to the diagnosis. I might go so far as to say that in many aberrant and abortive cases this forms the only guide, for without it many cases of sore throat, or of rashes in a scarlet fever epidemic, of nasal catarrh in an outbreak of measles or influenza, would go unrecognised. Next to epidemicity, the manner of onset in fairly typical cases gives the best clue to differential diagnosis. Thus they are all accompanied with pyrexia, which in —

Influenza Relapsing fever Scarlet fever Typhus Variola	Almost invariably comes on very rapidly, reaching its probable maximum within twenty-four hours.
Measles Pertussis Rötheln Typhoid	Comes on much less rapidly, taking two or three days or even longer (especially in typhoid) to reach its height.

After the onset a period elapses, nearly constant for each zymotic, before the further diagnostic point of the characteristic rash is available. We may utilise this period to draw attention to those symptoms which, if not actually the result of a temperature above the normal (a point made doubtful by many considerations), are at any rate the twin results with pyrexia of the toxæmias of disease. They are very important, as, when they are present, they suggest the continual use of the clinical thermometer; they also give the student a rational and firm basis for a written description of the symptoms of any

zymotic, of which indeed, with the rash, they form the bulk, stress being only laid on special points.

Following the plan laid down on p. 2, we have :—

THE SYMPTOMS OF PYREXIA

Alimentary System

Tongue.	Usually a tendency to get dry and dirty, in typhoid and in scarlet fever often typical.
Appetite.	Much diminished, probably digestion is nearly in abeyance.
Thirst.	Almost constantly increased.
Bowels.	Constipation the rule, in measles and typhoid diarrhœa not uncommon.
Respiratory and vascular symptoms.	The respiration and pulse are quickened, and in simple case in their ordinary ratio of 1 to $2\frac{1}{2}$. It is a sufficiently accurate and good clinical rule to expect an increase of about 10 beats per minute in the pulse rate for each degree F. that the temperature is over the normal. The principal exceptions are scarlet fever, in which the pulse rate is often accelerated out of all proportion, and influenza, in which there is very little acceleration. If the ratio of $2\frac{1}{2}$ to 1 is markedly altered, pulmonary or cardiac (endo- myo- or peri-cardial) complications must be strongly suspected and carefully looked for.
Urinary system.	The urine is usually diminished in quantity, increased in colour and specific gravity ; urates are very frequently deposited in excess on cooling. There seems to be no doubt that the toxins of all diseases are irritating to the kidney (by which channel they are largely excreted), hence the presence of a trace of albumen is very common ; it has little significance either as a signal of future kidney disease, or as a diagnostic point, except in scarlet fever, where obstinate nephritis is an only too frequent sequela, and in diphtheria, when albuminuria helps to decide the nature of an otherwise doubtful sore throat.
Skin.	Is often hot and dry, or may be moist and covered with sudamina.

Nervous system.	Headache, especially at back of eyes, a feeling of languor and debility with aching sensations in the limbs and back more or less severe, and the consequent restlessness, are all that can be ascribed to ordinary pyrexia ; should the temperature be over 105° delirium or coma may supervene, but these, with subsultus tendinum, floccitatio, etc., belong much more to poisoned than to heated blood, and may be seen with a subnormal temperature. It is worth while to note that small-pox, even in comparatively mild cases, is associated with a back-ache of quite disproportionate severity.

The characteristic rash of each disease now begins to appear, almost constantly as in the table below. With its appearance, though probably long before, the diagnosis will be complete.

Such are the usual features of the rashes of our zymotics, and they will, if anything like typical, be found sufficient to finally clinch a diagnosis which even for the most experienced may have remained doubtful previous to the appearance of the rash.

Desquamation.—Inasmuch as desquamation is usually considered very strong if not conclusive evidence in favour of a past infectious disease, it may not be out of place to state definitely once for all that it may be seen of almost any degree and character in patients who have been in bed for any disease. It is extremely copious in the course of some skin affections (dermatites), also after excessive doses of thyroid gland, and after prolonged (some days) bathing, as in typhoid ; but I have also seen it after pneumonia, in the course of kidney disease, as well as in patients affected with chronic spinal cord mischief.

We have now concluded the symptomatological diagnosis of our infectious diseases ; it simply remains to tabulate a few of the most salient features of contrast of the individual affections which are likely to be confounded, or which are of special interest to students.

[TABLE

TABLE REFERRED TO ON PAGE 36

Disease.	Date of Rash after Onset.	Locality.	Characters.
Varicella	Within 24 hours	Anywhere, frequently several crops of them.	Clear vesicles, not umbilicated, but they often leave scars.
Scarlet fever	Within 48 hours	Chest first, rapidly spreads to trunk, face, and limbs.	Bright, boiled-lobster colour, disappears on pressure (except in malignant cases), followed by large flaky desquamation, which continues longest on hands and feet.
Rötheln.[1]	About 3rd day	Trunk	Mottled like measles, or red like scarlet fever, followed by small branny desquamation.
Variola	3rd day	Forehead first, but soon nearly universal; only one crop, *v.* varicella	At first felt rather than seen as a soft, velvety feeling of skin with nodules under it; soon umbilicated vesicles, becoming pustules in 4 or 5 days, and almost invariably leaving scars.
Measles	4th day	Forehead first, and limbs, soon spreads to trunk	Mottled, patchy, does not absolutely disappear on pressure. Desquamation is small and branny.
Typhus	5th day	Wrists very early, soon universal. One crop only.	Mottled and hæmorrhagic spots, not disappearing on pressure, conspicuous.
Typhoid	7th or 8th day	Abdomen and back, successive crops	Small red, slightly elevated spots, disappearing on pressure, and usually want looking for.

[1] There is some dispute as to the existence of this disease; its clinical features, as allowed by the believers in it, are exactly intermediate between scarlet fever and measles.

	VARICELLA *v.*	VARIOLA
Fever and constitutional symptoms.	Scarcely marked, and even if pyrexia severe, still not much constitutional disturbance.	Usually very severe, and especially the back-ache.
Date of rash.	First day.	Anything in first two days will be preliminary atypical and probably hæmorrhagic; typical rash on third day.
Rash itself.	Often successive crops, non-umbilicated, hemispherical; practically never confluent; contents rarely more than milky; appear anywhere.	Always one crop only. Umbilicated and often confluent. Contents always purulent; forehead almost always the first.

It is but very rarely that any serious difficulty arises in the differential diagnosis of these two diseases, only an extraordinarily severe case of varicella could be mistaken for a mild attack of modified variola and *vice versa*. It is well to emphasise the fact (usually unnoticed or denied) that varicella does frequently leave pitted scars which may easily be mistaken individually for those left by smallpox, their greater number after the latter will be the best guide.

VARIOLA *v.* SYPHILIS

A far more difficult problem may occasionally arise to decide between the rash of variola and a varioliform syphilide. In both, the fever and constitutional symptoms may be very severe, and the actual spots may be identical in appearance, umbilicated and purulent. The most reliable point will be the situation of the rash, which in syphilis is nearly sure to be confined to the forehead, where it may be very copious; it is almost certain to be much more widely spread in variola. If doubt remains, a history of a chancre must be sought, and may clear up the diagnosis.

DIPHTHERIA *v.* "HOSPITAL," OR "ULCERATED' SORE THROAT

The following points are usually made in separating these troubles :—

Diphtheria.	Ulcerated Sore Throat.
Always derived from a previous case.	May arise *de novo* (at least without known exposure to previous throat case), from smell of drains, septic wounds, etc.
Temperature not high as a rule, 102° maximum.	Temperature higher, on an average 101° to 105°.
False membrane includes necrosed epithelium, if removed leaves a bleeding raw surface.	False membrane only an exudate, if removed does not leave a bleeding surface.
Albuminuria.	No albumen in urine.
Knee jerks often lost, and neuritis as sequela.	No loss of knee jerk or neuritic sequela.
Klebs-Loeffler bacillus found.	Other microbes but not the Klebs-Loeffler.

Notwithstanding the discovery of the Klebs-Loeffler bacillus, which must be by all admitted as the only cause of one specific form of disease called by the name of diphtheria, I am strongly convinced, as the result of personal experience, that there are several kinds of microbes which can give rise to forms of ulcerated sore throat, which by every *clinical* test are indistinguishable from the diphtheria produced by the Klebs-Loeffler bacillus; there may be sloughing and bleeding on removal of the slough, there may be albuminuria, and possibly disturbance of function of peripheral nerves—in fact, everything.

This statement may be accepted without its being held that I am preaching a dangerous doctrine, for I hold that the same care should be given to every case of ulcerated throat, and that the same isolation, especially as regards kissing and breathing the breath of the patient, should be practised, whatever the microbe may be, *for all such cases are undoubtedly infectious.* Nor do I disapprove of the use of antitoxin. I hold very strongly that if the throat be due to the Klebs-Loeffler bacillus, the injections will cure, when the case without them would be hopeless; and if the

throat be due to other microbes, the injections will certainly do no harm.

It may be that the acceptance of my view tends to vitiate the statistics of diphtheria, and hence those of antitoxin cures. This I cannot help, for I believe I am stating truth, and that even the most experienced will admit it. The confusion — if confusion there be—is due to the fact that we do not yet know all the powers for evil of all microbes, nor the laws which govern their growth on human tissues, and the powers of resistance of these same tissues.

We have already noted that rötheln as a distinct disease is not recognised by some observers. Accepting its existence, however, the following table, constructed from Dr. Roberts' *Medicine*, shows the principal characters of the three diseases — rötheln, measles, and scarlet fever—their likenesses and contrasts:—

MEASLES	RÖTHELN	SCARLET FEVER
	INCUBATION	
Eight to twelve days, never a few hours only.	Ten to twenty days, never a few hours only.	Two to five days, often within twenty-four hours.
	ONSET	
Fairly sudden, with rapid increment.	Sudden, but gets no worse.	Very sudden, acme in twelve hours or so.
	SYMPTOMS OF ONSET	
Catarrh of nose and eyes.	Sore throat, but not so bad as scarlet fever.	Throat symptoms very prominent.
	TEMPERATURE	
About 102°.	102° or lower.	102° or higher.
	RASH	
Appears about fourth day, distinctly to be felt, mottled patches,	Appears second day, papules brighter than in measles but	Appears in twenty-four hours, not raised nor to be felt, large

does not completely disappear on pressure, symptoms soon lessen when rash appears, rash lasts three or four days.	patchy, may coalesce and be bright like scarlet fever. Symptoms nearly gone when rash appears, rash lasts always four or five days, and may be eight or ten, longer than either of the other two.	areas, uniformly bright red; disappears entirely on pressure; symptoms worse if anything when rash is at its height; rash lasts four or five days.
PULSE		
Proportional to fever.	Proportional to fever.	Frequent, out of proportion to fever.
VARIETIES (only to be noted in Epidemics)		
Sine eruptione, sine catarrho, malignant (hæmorrhagic).	No marked varieties, but may occasionally be rather severe.	Sine eruptione, anginosa (throat specially bad), maligna (hæmorrhagic).
SEQUELÆ AND COMPLICATIONS		
Eyes, nose, and ears, but especially catarrh of air passages, also catarrh of intestinal tract. Cancrum oris.	None usually; very rarely nephritis.	Nephritis and dropsy, rheumatism, ear disease, endocarditis, cellulitis of neck, cancrum oris, abscesses.

Without the rash and without the presence of an epidemic to guide one, there are no features that will enable a diagnosis to be made between some cases of bad sore throat and the throat which may accompany either scarlet fever or (occasionally) measles.

Quite recently there have been described small whitish spots on the mucous membrane of the mouth and palate, which are said to be a pathognomonic indication of incipient measles.

TYPHOID *v.* TYPHUS

Typhus.	Typhoid.
1. Only seen in crowded poor communities; almost absolute contact required for infection.	1. Epidemics common in sparsely populated districts, conveyed by milk or water to a distance.

Typhus.	Typhoid.
2. Incubation, two to twelve days.	2. Very indefinite, may be a fortnight or more.
3. Onset very sudden.	3. Onset usually insidious, but may be rather sudden.
4. Symptoms markedly nervous and cerebral.	4. Symptoms markedly gastro-intestinal, though headache often severe.
5. Rash prominent to eye, as much on limbs as body, usually hæmorrhagic, appears earlier than in typhoid, five or six days.	5. Rash usually requires to be looked for; almost confined to trunk; rarely more than erythematous spots, entirely disappearing on pressure, rarely present in first week.
6. Constipation the rule.	6. Diarrhœa the rule, at any rate in second week.
7. Crisis almost invariable ending.	7. Lysis almost invariable ending.
8. Complications septic in character anywhere in the whole body.	8. Chief complications abdominal, — perforation, peritonitis, or hæmorrhage, but periosteal abscesses not very uncommon.
9. No characteristic lesions after death.	9. Characteristic lesions of intestines.

Considering the absolute contrasts that these two diseases offer, it would seem impossible that they should have been so long confounded; but we must remember that in those days typhus was very much more common than now, and therefore there were probably many cases aberrant from the type. If we remember, too, that what is now known as the "typhoid state" (dry, brown tongue, delirium, subsultus tendinum, and collapse) is the characteristic condition of any severe blood-poisoning of any nature, we may cease to wonder at the confusion.

The separation of the two is now of almost purely academic examinational (and historical) interest, for typhus is so rare that scarcely 1 per cent of medical men ever see a case.

The above table is quite sufficient should the differential diagnosis be required.

Weidal's test (the influence of the serum of a suspected person upon cultivations of typhoid bacilli) has recently been discovered. Its reliability would up to date seem to be of the very highest degree, in fact almost absolute if done by a skilled bacteriologist.

CHOLERA ASIATICA *v.* CHOLERA NOSTRAS (Severe Diarrhœa or Ptomaine Poisoning)

In England, luckily, we are not often called upon to deal with an epidemic of true Asiatic cholera; but, frequently enough, it is imported into our seaports, and occurs sporadically from this origin, so that the diagnosis is of some importance.

The essential etiological pathology of all such intestinal fluxes is the same. An irritant reaches the intestinal canal and causes a severe diarrhœa, to which, in the main, and not to the *specific* nature of the irritant, the symptoms are due. The diagnosis must then eventually rest upon the scientific discovery or proved absence of the comma bacillus of cholera, but the following clinical points are worth bearing in mind:—

1. If the outbreak or case be due to some ordinary irritant (chemical or living) in the food there will usually be an obvious history of such being the case, *e.g.* a public dinner, or all the members of a family being simultaneously attacked after a meal.

2. Children are almost the exclusive sufferers from our summer diarrhœa. If adults are also attacked, then paragraph (1) is nearly sure to be true.

3. In food-poisoning cases all the patients will be attacked so nearly simultaneously or under such circumstances as to exclude the possibility of infection from patient to patient.

4. Cholera nostras occurs with us almost exclusively in very hot summer weather, or in the autumn, when there is much spoiled fruit about.

5. Rice water stools are said to be very typical of true cholera, They are seldom passed by patients with ptomaine poisoning.

6. In a fatal case, with none of the above guiding indications, bacteriology must be called upon for the diagnosis.

ERYTHEMA SIMPLEX *v.* OTHER RASHES

To separate simple erythema from the erythematous rashes of more serious disease is a problem rather frequently arising in children. A new blanket, or underclothing of wool, a little dyspepsia, will frequently produce a rash closely resembling that of scarlet fever or erysipelas. In all cases of the slightest doubt an appeal must be at once made to the clinical thermometer, and again in twelve hours or so. Two or three observations on the tempera-

ture will clear up most cases. There is seldom pyrexia, at any rate of more than a few hours' duration, with the simpler troubles, and still less frequently an apyrexial condition when infectious disease is the cause of the rash. Besides the temperature the following points will be of assistance :—

Erythema.	Erysipelas, etc.
Margin of rash gradually fades into healthy colour. Locality erratic, no discoverable wound.	Outline of blush very distinct. Locality usually recognisable as that of a specific fever, or round a wound.
No constitutional disturbance, headache, vomiting, or chilliness.	Usually considerable disturbance of bodily health.
Probable history of some contact with irritating clothing material or known slight trouble.	Probable suspicion of infection.
Little tendency to spread after once it is out and discovered.	Usually spreads distinctly under observation, while symptoms remain or get worse.

INFLUENZA

Of late years we have had a repeatedly recurring recrudescence of this pest, and many opportunities for establishing its differential diagnosis. In the several epidemics it has almost seemed as though we had as many separate diseases to deal with—now an outbreak of contagious meningitis, now a pulmonary epidemic, and now one of gastro-intestinal catarrh or worse. In some a severe catarrh of the nose and eyes with a mottled rash has aroused suspicion of measles, while in others a severe tonsillitis and a bright red rash have made a diagnosis of scarlet fever seem almost justified.

The view which seems to me to best explain these vagaries is to assume that the microbe of influenza (it has not yet been isolated to the satisfaction and acceptance of everybody, though its existence is universally believed in) has but little specificity of its own. If it has a particular affinity it is for the nervous system, which it attacks with undue violence, causing disproportionate headache and pains, and followed by a debility out of all proportion to its pyrexia. Should the victim of its attack possess, however, a distinct *locus minoris resistentiae* it will attack that locus, and cause what is apparently a simple non-specific inflammatory attack, a pneumonia, a nephritis, a tonsillitis, a diarrhœa, etc.

As regards its actual diagnosis, my own experience would lead me to say that in cases uncomplicated by any actual inflammatory disease of an organ its main characteristics are:—

1. The absolute suddenness (even to the minute) of the onset of malaise and chilliness.

2. The presence of pyrexia, above 99.5 say, separates it from an ordinary cold in the head, especially in conjunction with—

3. The great intensity of the aching in the limbs and eyes.

4. During the first twenty-four to forty-eight hours the diagnosis, at least in children, must remain in doubt if any other epidemic is about.

5. At the end of this period, when we are expecting a rash or some other specific symptom, the temperature falls, and the patient is convalescent, unless some definite complication has occurred.

6. If such a local affection arise, the fact that it is due to influenza can only be determined by the prodromal symptoms which are absent in the simple cases.

In those cases where a rash and coryza or tonsillitis occur a certain and unquestionable diagnosis is at first impossible, unless there be a definite epidemic of influenza in the air, but we may remember:—

(*a*) The rash is more evanescent in influenza than in scarlet fever or measles.

(*b*) The whole febrile period without complications is also much shorter.

(*c*) Scarlet fever and measles but seldom occur more than once in a lifetime, and therefore a previous history of either is *pro tanto* a point in favour of influenza.

(*d*) Just the reverse holds of influenza, and a previous attack makes the present one more probably of that nature.

(*e*) In adults first attacks of scarlet fever and measles are comparative rarities, while influenza may have its first incidence at any age.

VENEREAL INFECTIOUS DISEASES

These include gonorrhœa, syphilis, and septic inoculations.

The diagnosis of gonorrhœa, with its special power of producing a suppurative discharge from mucous membranes, need not delay us. It is the truthfulness of the history more than the nature of the urethritis that requires consideration.

To differentiate a simply septic venereal sore from a true syphilitic chancre is much more difficult, and the fact that the

syphilitic and septic microbes are, with extraordinary frequency, inoculated together and grow side by side renders it often impossible to tell whether a given sore will ultimately prove syphilitic or not. Remembering, then, that many sores are both, the following points must be taken only as guides, not as infallible supports :—

Septic.	Syphilitic.
Incubation only a day or two.	Some three weeks. A sore (the date of inoculation for which can be sworn to) cannot be *purely* syphilitic if it appears within a week, but *it may be both.*
Suppuration and ulceration fairly free.	*Pure* syphilis does not suppurate, and probably not ulcerate ; but if a sore does ulcerate and become phagædenic, it is curious that it almost always contains also the syphilitic virus.
Frequently multiple, and can be inoculated on to a separate part of the body intentionally or accidentally.	Most usually single, and cannot be inoculated on to a distant part of the body.
Induration, if present, fades off gradually, and is only inflammatory œdema.	Induration is sharply defined like a wad let into the skin. Is something special to syphilis, not simple inflammatory.
Bubos often form, enlarge, soften, and abscess forms.	Bubos are hard and isolated, and if due *purely* to syphilis have no tendency to suppuration.
No rash, sore throat, or other constitutional disturbance at a later period.	Rash and sore throat coming on some weeks later are the only real pathognomonic features of true syphilis.

One cannot too strongly insist on the fact that a very large majority of venereal sores have the syphilitic virus in them, though its presence is for a long time obscured by the more active and rapidly-developing septic microbe, the work of which is soon prominent enough. The positive features of syphilis may later become most distinct, but a negative is proverbially difficult to prove, and nothing but six months from the date of inoculation will prove it satisfactorily. The question whether this is too heavy a price to pay for certainty belongs to the region of treatment, and cannot be discussed here.

CHAPTER IV

DISEASES OF THORACIC ORGANS

Section I.—The Lungs and Accessories

The principal symptoms arising from disease within the thorax are:—

(1) Pyrexia.
(2) Alterations in respiratory movements and rhythm.
(3) Alterations in cardiac sounds, position, and rhythm (*vide* Section II).
(4) Pain, and—
(5) Various other pressure effects.

The physical signs consist of alterations in the normal or average condition noticed on—

(1) Inspection.
(2) Palpation.
(3) Percussion.
(4) Auscultation.

I propose first to analyse some of these phenomena from the point of view of diagnosis, and then to offer a few remarks on the individual diseases producing them.

PYREXIA

This, *qua* thoracic disease, is only likely to be present as the result of inflammation of some thoracic organ, or produced by nervous reflex from irritation of the vagus, with or without interference with respiration or circulation.

Of the latter form there is but little in an elementary book to

say. It would certainly seem that hyperpyrexia might sometimes arise from this source, for it is seen occasionally in cases of pericarditis and pleurisy, and when rheumatic hyperpyrexia is accompanied by visible organic disease, this is almost constantly a pericarditis, so that arguments are not wanting to establish the position of a reflex hyperpyrexia through vagus irritation, though other factors, *e.g.* the rheumatic poison, may have a good deal to do with the temperature variations.

In cases of organic disease of the intrathoracic organs, on the other hand, the thermometer affords many useful points in differential diagnosis. In cases, for instance, where the physical signs tell us that bronchitis is present, it is a common rule of practice to consider that a temperature of 101° or over indicates the extension of the inflammatory process to some of the alveoli, so that bronchopneumonia is present, even though there be no tubular breathing, etc., suggestive of consolidation of lung, within the range of the ear. Again, we may suspect that an undoubtedly catarrhal area has been invaded by tubercle, or we may think of tubercle when a persistent cough exists without physical signs of a catarrh; here a nocturnal elevation of temperature with a normal reading in the day is a very strong piece of evidence in favour of the more serious condition, though it must be admitted that tubercle is sometimes apyrexial, and simple catarrh may be febrile even after its acute stage has passed. Lastly, in pleurisy with effusion, a nocturnal elevation of temperature, especially if associated with sweating, strongly suggests that the effusion is purulent, or, if proved clear, that it is due to concealed tubercle. The non-subsidence of a pyrexia after *apparent* removal of the cause, *e.g.* tapping, must be carefully watched to ascertain the meaning of the hitch in the proceedings; and similarly when a pyrexia, whose cause is known and whose course is usually constant, as in the common zymotics, does not follow the average rule, pulmonary complications must be suspected and carefully looked for.

It is important to remember that when breathing power is very seriously interfered with, as in some cases of pneumonia or bronchopneumonia, or in an ordinary case of bronchitis becoming capillary, the temperature may sink and become normal, or more probably subnormal, while the disease is making rapid strides to a fatal issue, so that a subnormal temperature in such cases is even more to be feared than pyrexia of moderate, or even severe degree, say up to 103.5 or 104. The same may be said in severe cardiac failure from any cause, though increased temperature is here less likely as

an initial phenomenon except in recent endocarditis, of which, indeed, pyrexia and a bruit are the chief indications.

In all cases of old or chronic lung and heart mischief, the thermometer is an important aid in determining whether the physical signs are produced by old changes in the tissues or by active processes still going on. If the former, there will most probably be no fever; if the latter, pyrexia may be present; at least if pyrexia is present, then the changes are probably active.

Alterations in Respiratory Rhythm

(*a*) *Cough*

Cough, as we know, is a peculiar modification of respiration which can be produced voluntarily, and can also often be suppressed by an effort of the will when the act would be attended by pain; but it is *essentially* a reflex act arising from irritation of nerve terminals or trunks, in direct or indirect communication with the respiratory centre, principally of the vagus or of the fifth nerve. It is designed primarily with the object of removing this irritant from the terminals of the pneumogastric in the air passages of the lung; but inasmuch as the respiratory centre is incapable of distinguishing between an irritant of terminals and one of nerve trunks, and between a removable and an irremovable irritant, the act is in many cases necessarily ineffectual. We are thus led naturally to a division of coughs into (*a*) coughs useful, (*b*) coughs useless, the former requiring to be helped, the latter to be suppressed as far as possible; and hence our first point in diagnosis is to try to separate the two classes.

First, as regards the sound and features of the cough itself:—

Cough Useful	*Cough Useless*
Moist, accompanied by a rattling or wheezing sound with each blast; if at first dry and ineffectual, ultimately results in the expulsion of some mucus or other material from the air passages more or less proportionate to the efforts made.	Dry, barking, or ringing cough, characteristically paroxysmal in many cases, *e.g.* whooping cough in later stages; always ineffectual in removing the essential cause of irritation. It may result in the bringing up of a small plug of mucus, but totally out of proportion to the efforts made.

N.B.—In both classes may be included a few unusual or rare cases of gross foreign bodies in the tubes, or of the membranous casts of plastic bronchitis, in each of which the cough may be dry, barky, and severe, or moist and wheezy, but may still result in the expulsion of the irritant.

Now as regards the clinical causation of the two groups. These we may tabulate as follows:—

Cough Useless

Cause.	Principal Diagnostic Points.
Disease of external ear.	The evidence will be chiefly negative, absence of other possible cause for a slight dry cough. Under such circumstances it has only to be remembered that a foreign body in the ear, or a little eczema, may cause such a cough for the diagnosis to be suggested, and the suggestion will lead to a careful examination of the part.
Elongated uvula.	Suspected by the cough and tickling in the throat being much worse (possibly only then noticeable) on lying down. Absence of thoracic signs of disease and presence of the long uvula complete the diagnosis.
Acute pharyngo-tonsillitis.	Cough (as opposed to mere laboured hawking of phlegm) slight, but attended with much pain, therefore often suppressed. Condition obvious on examination of mouth and pharynx.
Laryngitis, bronchitis, broncho-pneumonia, ordinary pneumonia, pleurisy.	In all these acute inflammatory conditions of the air passages the cough is, at least in the earlier stages,—say twenty-four to thirty-six hours—of the useless type, and may safely be mitigated with sedatives; the temperature, the history, and the painful cough are sufficient for a temporary diagnosis. For a complete one, *vide* under the appropriate subsections (pp. 97 *et seq.*).
Tuberculosis of larynx or of lung.	Both these forms of tubercle, in the stages of deposit, before ulceration or suppuration has occurred, are associated with a troublesome dry cough. In the former additional suspicion is aroused by hoarseness or loss of voice, and the laryngoscope will clear up the diagnosis. For a complete diagnosis of phthisis, *vide* pp. 84 *et seq.*

Cause.	Principal Diagnostic Points.
Malignant or syphilitic deposit in larynx before ulceration.	Cough and symptoms similar to tubercle, diagnosis made with laryngoscope. *N.B.*—In the later ulcerative stages of all these deposits in the larynx, though the cough may be in some degree useful, it is always persistent and distressing to a degree that renders some mitigation of it necessary.
Tumour pressing on pneumogastric or its branches, or on trachea or bronchi; aneurysm, enlarged glands, malignant growth.	Any of these tumours, in any form, may cause a dry, hacking, or brassy cough, which in itself should arouse suspicion of some such condition. Absence of other obvious cause, and especially if combined with paralysis of one or both cords, will be the chief diagnostic feature, but *vide* p. 105 for further indications.
Morbus cordis in early stages.	The back pressure of a leaking mitral will, on any extra exertion, cause a cough which—at first, at any rate—is distressing and useless. The diagnosis depends primarily on the presence of a bruit. Should this be absent and yet no other cause for a cough on exertion be found, the consideration of the heart sounds on p. 125 will be useful.

Cough Useful

As remarked above, cough may be looked upon as useful, and encouraged accordingly, when it removes irritating material from the air passages in something like proportion to its activity and force (the force may need reinforcement in the aged or debilitated). Hence we find it in—

The later stages of all acute inflammatory affections of the air passages and lungs when secretion has become excessive or suppuration established, or often enough when the disease has become chronic.

The later stages of phthisis when ulceration and breaking down of lung tissue has occurred.

In bronchiectasis, however arising.

In some cases of pleuritic effusion or of morbus cordis when it would appear that the air tubes are used as the means of carrying off excess of fluid.

In the bursting of abscesses, empyemata, etc., into the tubes.

The differential diagnosis of these conditions will be the object

of several sections in the succeeding pages. So far as the mere act of coughing is concerned, they offer no differentiating points. It is, however, worth while to draw attention to the suggestions given by the time of day at which a cough is worse. If it is worse *at night*, after the patient has got warm in bed, the suggestion is that it is due to some cause other than a mere mechanical stimulus or natural secretion, *i.e.* in an adult suggestive of phthisis or pneumonia, in a child of pertussis or bronchopneumonia. On the other hand, if the cough is worse *during the active working hours* or *on change of atmosphere*, this is suggestive of simple catarrh, as in mild bronchitis. It is possible that the explanation of this clinical fact may lie in a suggestion that the activity of the more virulent microbes is independent of the circumstances of the patient, whether active or quiet, asleep or awake, while the milder ones may be more dependent on respiratory activity, as we know natural secretions are.

The Sputum as an Aid to Diagnosis

Sputum is the natural sequel to a useful cough. As a guide to the naming of a disease it is not often by its naked-eye characters (its microscopical-bacteriological examination is most important) of great capital value, but as a guide to the improvement, or the reverse, of the condition of our patient's air tubes, during treatment, it is of very great significance. It is well to draw the student's and young practitioner's attention to the fact that the repeated physical examination of the chest which is allowable, and even necessary, in hospital work for teaching purposes, is not in private practice always advisable; and many patients will so strongly object to it that some other means must be adopted for estimating progress, and none are so useful as a few questions on the violence and frequency of the cough, and the amount of trouble experienced in getting up the phlegm, followed by an ocular and mental inspection of the sputum, which should always be kept for the purpose.

Phlegm, as the natural secretion or excretion of the mucous membrane of the air tubes, should be small in quantity, and consist merely of almost colourless mucus, with occasional cells and detritus of inhaled particles. It is what it should be, in country dwellers and non-smokers; but in towns, in smokers and others with dusty occupation, it becomes more or less coloured in agreement with these factors; hence black phlegm, unless we see it is due to dark blood, need not be a cause for alarm under such circumstances.

In disease the quantity is, as a rule, enormously increased, and much besides mucus is present.

Quantity.—Speaking of quantity only, quite apart from quality, it is important to note one point particularly, *i.e.* whether the excess is brought up in large mouthfuls or gulps at a time, with quiescence in the somewhat long intervals, or whether it arises from an increased frequency in the act of coughing, with but a moderate expectoration each time. The former indicates or suggests to us that there is a cavity or potential cavity of some kind communicating with a bronchus, and emptying itself by cough at intervals; such may be bronchiectasis, or abscess either of lung itself or neighbouring organ, or empyema. The latter tends to negative such a suspicion if roused by other methods of examination, and suggests instead a bronchitis, bronchopneumonia, or ordinary pneumonia, with excessive secretion, but without organic dilation of tubes; the absorption of a pleuritic effusion, or the presence of morbus cordis, will occasionally very much increase the quantity of sputum.

Quality.—As regards the quality of the sputum, the following are the principal pathological constituents to be found by clinical examination :—

Mucus (excess only is pathological),
Pus, and nummulation,
Blood,
Bile,
Lung tissue,
Hydatid hooklets and other parasitic detritus,
Microbes (particularly of tubercle and actinomycosis),
Fœtor,

on each of which I propose to make brief comments.

Mucus.—As this is the natural secretion of the air tubes, it follows that its excess or defect is the pathological phenomenon. In the early hours of a catarrh it is defective (hence cough useless and sedatives required). Later it becomes excessive but very sticky. This excessive stickiness is almost pathognomonic of pneumonia (cough still useless, or at least violent and out of proportion to result, and to be helped by liquefaction of phlegm). This indicates an acute bronchitis or pneumonia. Later still it becomes mixed with pus, and looser, indicating that the catarrh has caused suppurative processes which, unless very excessive, are known not to be of unfavourable omen.

Pus.—As mentioned above, this is found in the later stages of

all catarrhal affections of the bronchi, and hence, as a rule, is of little diagnostic significance; it causes mucus to assume a yellowish or green colouration. There are two conditions of suppurative sputum which it is important to bear in mind: (*a*) when the pus comes up very freely and almost pure, *i.e.* without much mucus, rousing suspicion of, or corroborating other indications of, the bursting of an abscess or empyema into an air tube, or the presence of bronchiectasis; (*b*) the condition known as nummulation of the sputum, in which small masses of pus float quite separately and isolated in the spittoon; it suggests the presence of small loculi where the pus can collect before being expectorated; it is seen in its most typical form in, and is very suggestive of, the later stages of tubercular destruction; it is found, but less frequently and typically, in pneumonia and bronchitis, especially if some of the smaller tubes have dilated.

Blood.—"Blood-spitting" is a very common complaint, and requires considerable care to ascertain its exact source, *vide* pp. 150 *et seq.* Suppose, however, we are satisfied that it arises from some pulmonary affection, we have still to consider its significance. It may appear in small isolated streaks in the mucus, or clotted and free, or it may be more intimately mixed with the sputum, imparting a uniform tinge to the excretion.

If of laryngeal origin.	Probably only in streaks or tiny clots, with pain in larynx and alteration of tone of voice. Unless other source obvious, a laryngeal examination must be made, and this will clear up the question.
If tracheal or acute bronchitis.	Again probably only in streaks and tiny clots; other signs of tracheitis and bronchitis.
In chronic bronchitis.	Often profuse, condition is fairly obvious in diagnosis, but the occurrence of hæmoptysis in the disease is frequently forgotten.
In phthisis.	Whether in an early or late stage the hæmorrhage may be smart, diagnosis must rest on other factors. Apices are especially likely to give added physical signs on deep inspiration. Bacillus, if found, is conclusive.
In pneumonia.	It is especially in pneumonia that the blood stains the sputum uniformly. Colour anything from mere rusty to dark prune-juice; the darker the worse the prognosis.
In morbus cordis.	Presence of bruits, or, more significantly, other signs of cardiac failure, *vide* p. 124.

In malignant disease.

The sputum of patients with recognisable malignant disease often contains small masses of blood-stained material likened to red-currant jelly; the point may be of importance in separating such from other consolidating lung trouble (*vide* p. 105).

Such are some of the main lines of thought towards a diagnosis in cases of pulmonary hæmorrhage, but it is easy to see that the bleeding itself or its character is of relatively small importance; other factors of much greater weight are involved.

Bile.—This is but a very rare constituent of sputum, but if present is practically pathognomonic of communication between an air tube and a pathological excavation of the liver.

Lung Tissue.—If fragments of elastic tissue are found in the expectoration (boiling with caustic potash and examining the detritus under the microscope is the simplest plan), the important caution is to be sure that what we see has actually come from the lung. It is not enough to find elastic fibres, which may have come from tiny fragments of food accidentally present; they must have the shape of a more or less open figure 8 indicating their origin from the bronchioles or alveoli. If such are present, they prove pulmonary ulceration to a demonstration, and render tuberculosis almost certain; doubt can only arise in some few cases of chronic disease of the lung, either fibroid pneumonia, or bronchitis with associated bronchiectasis. An acute gangrene will reveal itself by other more important features.

Hydatid Hooklets, or other parasitic detritus or eggs, are of course immediately pathognomonic of their source.

Microbes.—The bacilli of tubercle and other bacilli are of course equally pathognomonic; the only cautions are, first, to be sure that the staining processes have been properly carried out; and secondly, to remember the difficulty of proving a negative, and so excluding tuberculosis too summarily from the absence of bacilli in a few fields of the microscope.

Fœtor.—This proves that the microbes of putrefaction have not only reached the dead material of the sputum, but have had time to effect their peculiar changes in it; hence fœtor almost certainly indicates that the sputum has lain for some time in a potential cavity, *i.e.* a place where such changes could go on undisturbed, or that a piece of lung has itself died outright, or some foreign body is present in an air tube capable of putrefying; consequently careful search must be made for:—

(1) Bronchiectasis.	Usually associated with chronic bronchitis. Phthisical cavities are curiously almost exempt from this putrefactive process, though their contents have a peculiar mawkish smell very characteristic.
(2) Abscess of lung. Intrinsic or bursting into lung from within.	The main diagnostic features of intrinsic abscess are (*a*) fœtid expectoration, which (*b*) consists of nearly pure pus, with putrefying detritus, and (*c*) the discovery over a localised area of the lung of pathological physical signs (*vide* pp. 64 *et seq.*). Abscess arising elsewhere and bursting into the lung will have revealed itself before rupture probably. The fœtor (if present) and coughing up of quantities of pus merely indicate that communication has been established with the air tubes.
(3) Gangrene.	The fœtor of pulmonary gangrene is utterly indescribable; once smelt it cannot be forgotten, and when occurring with symptoms of serious blood-poisoning leaves no room for doubt or error.

It is well to insert here a caution against mistaking foul breath for fœtor of sputum; the latter is only one cause of the former, and that a rare one, the nose, mouth, and stomach being far and away the most frequent sources of foul breath.

To sum up the diagnostic value of an examination of the sputum, we may say that nearly every disease of the respiratory apparatus has, or may have, a form of expectoration which is fairly characteristic which, at any rate, lends strong corroboration to other physical signs and symptoms, and at times takes a leading position as an indicator of the essential condition of the organ—*improvement or the reverse*—under treatment.

Besides cough and its attendant expectoration, there are one or two general alterations in respiratory movements which deserve a brief mention and analysis for diagnosis.

Generally accelerated Respiration.—This, apart from intentional or voluntary quickening of the movement, essentially means that the supply of oxygen to the tissues is insufficient for their immediate (it may be) temporary needs. This may be analysed into:—

A. Conditions in which an extra supply is required.

(*a*) Exertion.	A purely healthy and physiological condition, exaggerated in convalescence when the tissues

	are rebuilding their stable capital of nutritional material.
(*b*) Pyrexia arising from any disease.	Oxidation is quickened throughout the body. I have already (p. 35) noticed the importance of the temperature-respiration-ratio in drawing attention to pulmonary complications when other complaints of lung trouble by the patient are absent. This rule is particularly valuable in the specific fevers, when cough may be deliberately suppressed because of the pain it causes, or when the patient is too weak to cough.

B. Conditions in which increased frequency of respiratory movement is required to keep up the ordinary normal supply.

(*a*) Diminished aerating surface in lungs.	Seen in bronchitis, pneumonia, emphysema, collapse of lung, phthisis, etc.; in fact, any disease filling the air tubes or destroying alveoli.
(*b*) Diminished or obstructed circulation of blood through the lungs.	Seen in valvular or muscular disease of the heart, in emphysema, etc.

N.B.—It is probable that these two causes never act absolutely independently. Pulmonary disease in itself, *e.g.* emphysema, invariably tends to obstruct the circulation, and cardiac disease tends to fill the air tubes.

(*c*) Poverty of blood in oxygen carriers.	Seen in anæmia, from whatever cause arising.

From the variety and number of the diseases and conditions in which increased frequency of respiration occurs, it will be seen that the act is in itself not of very great value in differential diagnosis, but there are one or two points in it well worth attention:—

In chronic disease of the lung, *e.g.* phthisis or bronchitis, the patient and his tissues, especially if much wasted, may have got so accustomed to the smaller supply of oxygen that shortness of breath *will not be complained of, and may even be denied*, until some extra exertion is called for; whereas in acute disease of the lung the shortness of breath is at once obvious, and often very distressing.

In old people, whose chemical changes are not extremely active, very serious lung and pleural trouble may come on extremely insidiously without any rise of temperature or general discomfort on

the part of the patient, except for a little shortness of breath; hence it is, in such patients, very important to inquire specifically for such complaint, and make a careful physical examination of the chest on the slightest suspicion.

In young babies, again, and infants of feeble vitality, slightly quickened respiration (it must not be forgotten that their normal respiration rate is from 25 to 35), accompanied by a little wheezing, may be all the evidence of a bronchitis that is rapidly proceeding to become a dangerous and even fatal illness.

Cheyne-Stokes breathing is a curious variety of respiratory movement, in which the patient passes gradually from a condition of apnœa (which may have lasted some seconds) through a period of steadily increasing frequency up to one of very great frequency of respiration. It is seen now and again in almost every form of disease — uræmia, chronic nervous disease, concussion of brain, etc.; its morbid physiology is unknown, and it has no clinical *diagnostic* value; but it is of very grave *prognostic* significance, few recovering when the condition is well marked as an added phenomenon to the symptoms already present indicative of acute disease.

Dyspnœa is a term worth defining, for I find that students so frequently use it as the scientific equivalent of shortness of breath. It really means difficulty of breathing; no patient ever yet complained of "dyspnœa," but many complain, and that not indifferently, of "shortness of breath," or of "difficulty in breathing," terms which are sufficiently explicit in themselves, and should not be confounded. My own teaching and practice is to reserve the term dyspnœa either for those cases in which the patient complains of difficulty in breathing, as, for example, in most cases of asthma, or in which we have strong objective evidence of a mechanical interference with the free entrance of air to the lungs, *e.g.* diphtheritic membrane, spasm or paralysis of larynx, pressure on the trachea, etc. Thus used, the term is of some diagnostic use, indicating one of the above conditions as opposed to the numberless causes of "shortness of breath."

Diaphragmatic Breathing. — This is more or less a natural phenomenon, in the male sex at any rate, but may become grossly exaggerated, and assumes then very grave prognostic significance. My own experience of it would compel me to say that it has two diagnostic suggestions: (1) that the *nervous* mechanism of respiration is gravely interfered with either by gross disease in the thorax, or more probably by a serious lesion of the nervous system; (2)

(a negative inference) that there is no accumulation of fluid in the pleural cavity.

Pressure Effects

The thorax is certainly not an incompressible cavity like the cranium; its roof, consisting of the dome of the pleura and the cervical connective tissue and other soft structures, and its floor, viz. the diaphragm, are certainly both easily capable of displacement or extension; but the side walls, consisting of sternum, ribs, and vertebræ, are comparatively inextensible, and the individual organs themselves, except the bulk of the heart, are so firmly fixed in their places by the mediastinal tissue, that no very great addition can be made to the bulk of an intrathoracic organ, and no neoplasm can attain any great size without leading to compression of some important structure, or to manifest displacement of the heart. The gross effects of the pressure or displacement are readily enough recognised in most cases, but to estimate exactly the diagnostic value of these various pressure effects requires a considerable knowledge of morbid-anatomical possibilities and of the relative positions of the several structures in the thorax. Into these points I do not propose to enter fully, but I wish to give an analysis of the principles for completing a diagnosis, leaving the application of these principles to be worked out in individual cases.

We require first, then, a list of classes of structures that may be compressed or eroded and destroyed. These comprise:—

1. Hollow vessels:—	Principal Results.
(*a*) Lymphatics and thoracic ducts.	Œdema of chest wall or arm. If the thoracic duct is blocked, chylous ascites is very possibly present.
(*b*) Veins.	Œdema, as in lymphatic block, but the superficial veins of the district involved are probably also enlarged.
(*c*) Arteries.	Alteration in the time and volume of the corresponding pulse felt in an accessible situation.
(*d*) Air tubes and lungs.	Cough with little expectoration, shortness of breath, inspiratory dyspnœa if large tube compressed, possibly collapse of lung.
(*e*) Heart.	Displacement of apex beat, possibly unusual bruits and altered rhythm.
(*f*) Œsophagus.	Difficulty in swallowing, or rather feeling of food sticking and not reaching the stomach, possibly regurgitation.

2. Nerves :—	Principal Results.
Of which the most important are :—	
(*a*) Recurrent laryngeal.	Spasm or paralysis of vocal cord, hoarseness or alteration of voice, possibly dyspnœa and ineffectual cough.
(*b*) Phrenic.	Hiccough or paralysis of diaphragm.
(*c*) Pneumogastric and cardiac plexuses.	Disturbance of heart beat, slowing, quickening, or irregularity, possibly vomiting.
(*d*) Lowest cord of brachial plexus.	Disturbance of function of ulnar and other nerves of arm.

The nature of the nerve disturbances will be better understood by reference to p. 315.

3. Bones—vertebræ, ribs, or sternum.	Severe pain in back, worse at night (this is a very characteristic feature of bone pain, whatever bone be affected and whatever the cause), possibly felt all round the chest from implication of intercostal nerves.
	Local pain, and possibly erosion with bulging.
4. Roof.	Visible bulging above clavicle, either persistent or on coughing.
5. Floor.	Displacement with easy palpation of liver below the ribs, loss of free respiratory movements of diaphragm.

These are the principal results of pressure. The chief causes may be thus tabulated :—

1. Serious deformity of chest from old spinal caries, severe rickets, osteomalacia, paralysis, etc.	The deformity is obvious on inspection and palpation.

N.B.—In a chest seriously deformed by old or recent disease, it must be borne in mind that even a slight attack of pulmonary disease—pneumonia, bronchitis, etc.—is very apt to prove serious or even fatal, owing to the great curtailment of breathing room. This is merely an illustration of the great principle of the loss of adaptability for emergencies or extra work entailed by old structural damage, a principle easily remembered in the case of kidneys,

livers, etc., but apt to be forgotten for more obvious bodily deformities.

2. Aneurysm.	Especially apt to exert pressure on recurrent laryngeal and other nerves, trachea, left innominate vein, and on bone, but *vide* p. 107 for further details.
3. Solid growths.	Press especially on veins (intercostal, azygos, etc.); *vide* p. 107.
4. Pleuritic and pericardial effusion.	Cause displacement of heart and apex beat particularly, but *vide* p. 102.

Pain in Chest

Before considering the differential value of a pain in the chest it is well to locate the unhappy feeling rather exactly, because many people seem to be unaware of the thoracic boundaries, and call the abdomen the chest, either out of such ignorance or out of modesty. Even when we have located the pain with accuracy, it is not always easy to at once fix the offending organ, for some abdominal troubles, notoriously dyspepsia (*vide* below), may cause pain referred to the chest, and I have known pleurisy cause such extreme abdominal pain as to lead to an erroneous diagnosis of peritonitis.

Chest pain, if due to organic disease situated within the thorax, is almost invariably due to one of the following:—

Traumatism.	History obvious.
Spinal caries.	Pain of girdle character and vertebral spines tender, possibly also deformed in position.
Aneurysm.	May simulate caries in pain; *vide* p. 107.
Pleurisy.	Much worse on deep inspiration, especially on coughing; rub conclusive.
Pericarditis.	Localised in precordial area; heart beats unusually frequent; rub conclusive, but *vide* p. 127.
Heart disease, especially aortic.	Bruits heard; pain usually anginal, *i.e.* very severe tight cramping pain, sudden in onset and probably caused by some exertion, though this may be only slight.

Hence we see that even a superficial physical examination is not likely to leave us long in doubt about the pain of acute peritonitis, calculus, or other gross organic disease of abdominal viscera. If the physical signs in the chest are not marked, those in the

abdomen are only too obvious. But the relation between cause and effect is by no means so easy to trace in the case of pain in the chest arising from functional dyspepsia, as mentioned above. Such a common correlation is there between heart and stomach that it is now a commonplace of medicine to say, "If a patient complains of his heart, the first suggestion is that his stomach is out of order; if he complains of his stomach, don't let him go without examining his heart carefully." A few leading questions on the relationship of the pain to food, to movement, to respiration, will, however, tend to clear matters up; the following points may be taken as guides :—

PAIN IN HEART REGION

If of Gastric Origin.	If really Cardiac.
Appears after food, and apparently as the direct consequence of its ingestion.	Appears also quite irrespective of whether food be taken or not.
Accompanied by feeling of fulness in stomach, often relieved momentarily by belching.	No such feeling of fulness, not relieved by eructation of wind.
Not increased by walking, which is easily possible.	Increased by active movement, which may even be impossible owing to the severity of the pain.
Heart sounds normal in rhythm and character.	Probably some cardiac bruit present, or at least alteration in rhythm and volume of pulse from the normal.

Notwithstanding these differences, we must not forget that the two troubles may coexist; that, in fact, the heart trouble may be producing the dyspepsia, and *vice versa*, the dyspepsia may produce an irritable heart. We must then leave the history and other indications to decide which is cause and which effect, a decision all the more important as treatment will be founded on it. If the heart mischief is primary, there are almost certain to be other indications of it—some shortness of breath, œdema of legs, etc.; it is very improbable that dyspepsia will be the only symptom; *per contra*, if dyspepsia is the primary trouble, there will almost certainly be a history of discomfort after meals long antedating the pain in the heart.

There is still one form of pain in the chest which requires

attention from a diagnostic point of view. It is very common in my experience, situated about the lower ribs on either side, but very much more common on the left. It derives its interest from the entire absence of any physical sign to account for it; no heart disease, no friction sounds, tongue clean, bowels regular, and no discomfort after meals. A stitch in the side from excessive exertion or laughter, etc., is a common phenomenon, due probably to cramp of one or two intercostal or abdominal muscles or segments; intercostal neuralgia, too, of a typical darting character I have heard of and read of, but seldom seen: but the pain to which I refer does not agree with either of these. It is constantly present more or less, but is much increased by coughing or violent respiratory movements, and remains fixed in its original situation. I do not say that I can point to the cause of all such pains, but I would draw attention to what I believe to be the explanation of many of the cases.

Nothing is commoner on post-mortem examination than to find, besides the definite disease which has caused death, one or more of the following distinctly pathological conditions, evidence of past local trouble, viz.:—

1. Adhesions of lung to diaphragm.	Diaphragmatic pleurisy.
2. Adhesions of spleen to diaphragm.	Perisplenitis.
3. Adhesions of spleen to intestine.	Perisplenitis.
4. Adhesions of intestine to gall bladder and liver.	Local peritonitis.
5. Adhesions of liver to diaphragm.	Perihepatitis.
6. Thickenings of splenic capsule.	Fibrous and even calcareous.

The argument in regard to such post-mortem findings runs thus:—

1. They are definite and indisputable evidence of old local trouble.

2. They offer no suggestion that the trouble was of a disabling character, or so severe as to necessitate, or perhaps even suggest, confinement to bed; and hence they are easily forgotten afterwards.

3. When recent, they were in all probability associated with pain, and even as old pathological adhesions they probably cause

pain by restriction of the natural movement, which would follow ordinary energising of abdominal and thoracic muscles, *i.e.* attempted movement.

4. The pain would probably be fixed in position about the lower ribs, quite likely be made worse by extra movement, as of cough, or even by the digestive movements or physiological congestion of the stomach and intestine—in fact, just such a pain as we are discussing.

5. From their position, and from the little movement-in-contact of organs concerned, they would be unlikely to give rise to an audible rub or other objective physical sign.

On the other hand, I am obliged to state that I am unable to say definitely that these persons had suffered from our present form of pain, nor can I say that they had not a definite disabling illness which might have resulted in such adhesions and thickenings. On the whole, I am inclined to think that the appearances are too common in proportion to histories of severe illnesses, and that they do explain most satisfactorily the pain under discussion.

PHYSICAL SIGNS OF THORACIC DISEASE

As with the symptoms, so with the physical signs of intra-thoracic disease. I do not propose to go systematically through each and all, but merely to touch on those points which are of special diagnostic value, or on which, experience in teaching has shown me, the ordinary text-books hardly lay sufficient stress.

Comparison of the Two Sides

It cannot be too much insisted upon that the thorax in each and every individual is a closed box—a drum, in fact—with its own amount of external covering or damper of vibration, its own individual shape and build—in fine, its own complete group of special peculiarities; consequently it is impossible to lay down any law as to what should constitute its normal shape, or the normal sound elicited by striking it. There is no such thing as an absolute guide to discriminate the normal from the abnormal; that which would be natural in the chest of a healthy, stout woman, or in a baby, or even in an old deformed chest, would be distinctly pathological in the chest of a thin man, an adult, or in a well-formed chest. Our only reliable guide, then, is *a comparison of the two*

sides of the same chest, combined with experience of average chests; and be it remembered that the two sides must be examined and compared at corresponding points and in a similar manner. It is no good comparing any of the physical signs, seen, felt, or heard, of the front or axillary region of one side with those of the back or suprascapular region of the other, nor is it any good to lay the plexímeter finger along the ribs and spaces in one case, and across them in another. Thus compared, a little swelling or œdema on one side only, a little difference in movement, in tactile vocal fremitus, in dulness on percussion, and especially in loudness and distinctness of breath and voice sounds acquires an enormous increase in importance from what it would have if the (possibly) pathological change were found on both sides to the same degree. In one case disease past or present is there; in the other we are probably dealing with a natural, if not healthy (as in old deformity), condition. *Never examine one side of a chest only, however obtrusive be the local complaint of the patient; compare, compare, compare! !*

Physical Signs give Physical Conditions only

This limitation in the diagnostic utility of the physical examination of the chest is too apt to be lost sight of, and deserves emphasis. It enables us to see a bulging and motionless chest, but it does not decide the reason for this. It proves the presence of undue secretion in the air tubes, but it does not give us the tubercle bacilli which may be causing it. Dulness on percussion does not decide between serum, blood, and pus; and external signs of internal pressure leave aneurysm or malignant growth still in doubt. Bearing this limitation in mind, we may proceed to consider what can be learnt by it.

Inspection

The principal points noticed on inspection, and their preliminary indications, may be thus sketched out:—

Old or permanent deformity. Passed rickets.	Probably symmetrical, and causing forward projection of sternum, or, *per contra*, considerable depression of sternum and costal cartilages.
Old spinal curvature.	Probably asymmetrical, hump on back, and twisting on vertebral axis.
Emphysema.	Rounded, barrel-shaped chest, with but little respiratory movement.

N.B.—All three will possibly, and even almost certainly, materially alter the results of percussion and auscultation, owing to changes of position in the organs and alteration in lung structure.

Recent bulging of one side.	Suggests a large accumulation of fluid in pleura, or a new growth. Measure the two sides to see if the increased size of one is real, or apparent only. Look for œdema of one side or anything like a local bulging or pointing.
Fat or thin chest.	Remember fat is a bad conductor of any vibrations; the condition is obvious enough in itself, but must be remembered in listening to or percussing the chest.
Enlarged veins.	An important sign of obstruction to the venous return of blood. If due to cardiac disease or to generalised lung mischief, it will be noticed chiefly in the neck, jugulars, etc.; if due to a local pressure (aneurysm or growth), more likely in the walls of the chest, or arms, and, possibly, abdomen.
Œdema.	If confined to one arm or one side of the chest, it is very strongly suggestive of a malignant growth with much local pressure. If general, more likely to be only part of a renal or cardiac dropsy.
Local tumour.	If only connected with skin or muscles, belongs to the domain of surgery. If evidently coming from within the thorax, may be a malignant growth, an aneurysm, or a pointing empyema.

Palpation

By feeling the chest we shall perceive:—

Œdema.	Already seen by inspection, but its limits are best ascertained by the present method of examination.
Tactile vocal fremitus is increased by anything which allows of a freer vibration of the chest wall in response to the vibrations of the voice, hence noted in—	(*a*) Thin chests, or in parts of the chest least covered. (*b*) More powerful voice of a man, and in deeper tones. (*c*) Consolidation of the lung, of whatever nature, *provided that it still remains in immediate contact with the chest wall.*

	(*d*) Occasionally by very large effusions into the pleura completely compressing the whole lung.
Is diminished, *per contra*, by anything that damps off the vibrations.	(*a*) Fat chests and parts well covered either by fat or muscle. (*b*) Less powerful voice vibrations in women and children, and higher tones. (*c*) Separation of the lung from the chest wall by fluid collections. (*d*) Growths of parietal pleura not involving visceral layer or lung.
Position of heart's apex beat.	*Vide* pp. 123 and 133. Not felt in fat people very often, nor in emphysema. Fluid effusions in pleura are the great cause of displacement.
Thrill of heart beat.	*Vide* p. 114.

Percussion

By percussion we estimate the relative degrees of resiliency or elasticity of the chest wall, and get a note varying in tone and quality from hyper-resonance to complete dulness.

Resiliency or elasticity of wall can only be properly appreciated when the finger is used as the pleximeter (hence the absolute objection to the use of all artificial pleximeters of any material), and a knowledge of its varying degrees in deformed chests and in health, its absence or presence with associated signs in disease, can only be gained by experience; it cannot be communicated by writing, but once acquired its value is very great. We may classify its essential features as under, with their indications:—

Its complete presence with ordinary but varying degrees of resonance.	Indicates that the parietal pleura is practically healthy, free from any degree of deposit or growth, and not thickened to any great extent, though it may be adherent to the visceral layer; and that there is no extensive pleural effusion. Pneumothorax, emphysema, or bronchitis and other *lung* conditions are not thereby excluded.
Its presence with a note known as dull gives an im-	This is found in pneumonia and other conditions of consolidation of the lung, *provided that it is not separated from the chest wall by fluid, and*

pression as though one were percussing a thick piece of wood—a wooden tone.	*that the parietal pleura is not much implicated.* This is the special utility of the phenomenon, viz. to decide as to whether a pneumonia is or is not accompanied by much pleural effusion. The exploring needle is the only other method of determining the point.
Its absence with a note known as dull gives an impression as though one were percussing a brick wall or the thigh, and renders the dulness absolute.	This indicates a pleural effusion of considerable extent, or a marked thickening of the pleura, but—*N.B.—It does not give the reason for the effusion or thickening, a new growth, recent inflammation, or long antecedent disease.*

It will be noticed that these indications are directed almost entirely to the condition of the pleura, or to the introduction of considerable consolidation of the lung. The reason for this is in general terms easy to see, viz. the elasticity, and, I may say, the note elicited, depend entirely on the capability of the part struck to vibrate. This capability is fixed primarily by the shape (including old deformity now become the original shape), structure, etc., of the chest (*vide supra*), and is diminished (or increased) more by an alteration in the pleura and its contents, *e.g.* fluid (or air-increase) and introduction of considerable underlying solidification, than by anything else.

Note elicited apart from Resiliency of Wall.—To explain accurately the variations in this note is not so easy as the explanation of the variations in general elasticity of the chest wall, but it seems fairly obvious that if a certain combination of healthy lung, pleura, and other structures, including the chest wall, gives in response to a blow a certain note, then an alteration in any of these organs or conditions should alter the note *at the spot where the alteration exists.* If this be not an entirely satisfactory scientific explanation, it is, at any rate, the expression of a clinical fact learnt by experience; and hence the incalculable importance of comparing the two sides of the chest for detecting mischief *on one side only.* We must not forget, however, in this comparison, the asymmetrical position of the organs in the chest, the heart on the left, the liver below on the right, and the peculiar course of the aorta, with asymmetry of its main trunks. It is not worth while to attempt to tabulate the clinical causes that may produce alterations in the percussion note, but attention may

be drawn to the paragraphs on the diagnosis of early phthisis, pneumothorax, excavation of lung, etc.

Auscultation

By the application of the ear (immediate) or of some form of stethoscope (mediate auscultation) to the chest, we try to appreciate, and then estimate the meaning of, any sounds which are produced by the natural movements of respiration and circulation; or we apply artificial means such as tapping or shaking for the same purposes. These sounds may be subdivided in their origin into:—

A. Those arising in and transmitted through the air tubes, including—

(1) The natural sounds of the passage of the respired air.
(2) Adventitious, or sounds added on to these natural ones, but still simply respiratory in causation.
(3) The sounds produced by the voice.

B. Those arising from pathological conditions of the pleura:—

Friction sounds.
Creaking.

C. Sounds produced artificially by tapping the chest, and succussion.

D. Produced by the circulatory apparatus. *Vide* subsequent section.

As regards their characters and meanings, we may classify them as follows:—

A. (1) *Natural Breath Sounds*

Point where heard.	Normal sounds.	Significance.
Over larynx and trachea.	Inspiration and expiration, distinctly a noisy, non-musical rush of air, and both equally so = tracheal breathing.	In laryngeal obstruction stridor and hoarseness may be detected.

Point where heard.	Normal sounds.	Significance.
Over larger bronchi, *e.g.* interscapular space or R. sterno clavicular articulation.	Inspiration louder than expiration, but the latter very distinct and audible = bronchial or tubular breathing.	Has no abnormality in itself, only becomes abnormal by— (*a*) Being heard in the wrong place = too free conduction through solid lung or dilated tube. (*b*) Not being heard in the right place from compression of a tube, etc.
Over ordinary lung tissue.	Inspiration distinct, ending in or constituting vesicular murmur; expiration scarcely or not at all heard.	Its absence is certainly abnormal, *e.g.* over lung solidified by any cause. In babies, in unusually thin adults, and in one lung which is doing the work of two, the vesicular murmur is undoubtedly exaggerated, and constitutes the harsh breathing of some authors.[1]

(2) *Additional or Adventitious Sounds* are in their nature essentially pathological, so we have only to discuss their variations and meaning. They may be subdivided as follows:—

I. Interrupted:[2]

1. Fine sounds (crepitations) are such as would be produced by the separation of two sticky surfaces, that

[1] Some authorities on auscultation will not admit the use of the term "harsh breathing," but I must say I know of no other term that can better describe the sounds heard in the three conditions mentioned above.

[2] By thus strictly defining the terms "crepitations" and "rales" according to the time in the respiratory cycle in which they are heard, it seems to me that they gain in accuracy for use, and indeed also in meaning and significance. The adjectives "coarse" and "fine" may still be used for either, occurring in its proper time; and from the coarseness or the reverse, and from the time definition, we may form a tolerably accurate opinion of either (1) the probable size of the tube in which the sound arises; (2) whether the air has an actual thoroughfare through the tube, or only goes into and back again out of it; (3) the state of the secretion, the more liquid, the coarser the

is, in fact, the actual cause of them, and their place of origin is the alveoli and very finest air tubes of the lung. *They are heard during inspiration only.*

2. Coarser sounds and moister (rales) are produced by the passage of air through a more or less viscid secretion, and represent the bursting of air bubbles in the fluid; they are produced in all tubes larger than those causing crepitations only by the passage of air through the secretion. *They are heard therefore during both expiration and inspiration.*

II. Continuous sounds:

These are the equivalent of the term rhonchus as used by the late Dr. Fagge, the main point of the definition consisting in the sound remaining at a certain level or pitch, possessing, in fact, a certain musical tone and quality throughout inspiration or expiration, or both. Of the sounds themselves there are many varieties: whistling, cooing, snoring, etc., according to the depth of tone or originality of term-invention of the listener. They are produced by secretion adhering in local masses to the wall of a larger tube, thus forming a sudden and adventitious narrowing of the lumen round which the air passes with a musical note.

(3) *Voice Sounds.* (For Tactile Vocal Fremitus *vide* under Palpation.)

	Normally.	Abnormally.
Over trachea and larynx.	Loud and fairly distinct.	Hoarseness the only detectable variation, owing to irregular growths or secretion covering the cords.
Over larger bronchi.	Not so loud, and more indistinct.	No idiopathic abnormality, only abnormal like the breath sounds by heterotopy.

sound, and the more likely, in the finer tubes, to allow air to actually bubble through it both ways. The two may both occur in the same breath consecutively to one another, a circumstance which would strongly suggest that an alveolar or capillary affection were supervening on a catarrh of the larger tubes, a most useful danger signal, therefore.

	Normally.	Abnormally.
Over lung tissue.	Mere confused murmur or not at all.	Increase of spoken words (bronchophony) denotes a pathological, increased facility for transmission from the larynx (solidification of structure, dilated or ruptured tube, cavity, etc.). Increase in whispered words (pectoriloquy) denotes still greater facilities for sound transmission from the larynx. Altered sound of voice, only one noticeable form (ægophony), a peculiar bleating tone strongly characteristic, if not pathognomonic, of pleural effusion.

In addition to the above classes of regular voice and breath sounds, we must notice one or two important modifications of them.

Cavernous Breathing is primarily an exaggeration in loudness of tubular breathing, but it has in addition a musical character of its own—comparable to the notes produced by blowing over the mouth of an empty bottle, and due to the same essential cause, viz. interfering currents of air echoing in a hollow. When heard in typical form this cavernous breathing is characteristic of a cavity, pulmonary or pleural, communicating freely with a tolerably large bronchial tube.

Tinkling Sounds produced by the falling of drops into the body of a liquid contained in a resonating chamber; hence they are only heard when the liquid has been violently driven over the surface of either a pulmonary or pleural cavity by coughing or succussion.

Cogwheel Respiration is a peculiar form of interrupted inspiration, as though the air entered in a series of little jerks. It is almost confined in its appearance to the upper lobes of the lungs, and was formerly thought to have much, now only a very little, significance

as indicating early stages of phthisis, causing unequal difficulty of air entrance into certain lobules. My own experience would lead me to say that it is frequently met with in perfectly healthy lungs, and that its chief significance is a very minor one, viz. that the patient is nervous, and totally unaccustomed to breathe by intentional or voluntary movements.

B. *Pleural Friction Sounds* are well known, to-and-fro rubbing sounds. There are only two sources of difficulty in their diagnosis: the one is to separate them from fine crepitations, and the other from pericardial friction; the latter is identical in character. From the former they are separable by being coarser, and sounding more immediately in contact with the stethoscope, but principally and essentially by being heard both with expiration and inspiration; a doubtful sound heard with both movements must be either a rub or rales, and the latter will be evidently bubbling. The need for separating them from pericardial friction sounds arises only in the neighbourhood of the pericardium, and there the simple manœuvre of listening while the patient holds his breath will usually be sufficient, pleural sounds then of course ceasing, except in the occasional cases when pleuro-pericardial adhesions exist, and then the important diagnostic point is to recognise by the continuance of the sound during the cardiac cycle that the pericardium is affected.

Pleural Creeks are rare, and only heard in exceptional cases of very old-standing chronic dry pleurisy.

C. *Sounds heard by Tapping the Chest and by Succussion.*—These include:—

Bell Sound, the ringing echo across a chest produced by laying one coin on the wall and striking this with another coin. The necessary condition for its production is a fairly large empty space, and hence it is useful in detecting a pneumothorax, and its presence may be corroborative of other evidence of a pulmonary cavity.

Splashing Sounds produced by shaking the patient; can be but seldom required, but indicate, when heard, air and fluid in the pleura.

This classification gives in a fairly comprehensive and comprehensible manner the chief ordinary pathological sounds heard in the thorax. For these abnormalities of natural phenomena my chief aim has been to indicate the principles and reasons which, when well grasped, will give a rational foundation for explaining any others that may be found in a given case. In practice the difficulty is not so much to hear and understand the physical meaning of

intrathoracic sounds, as to know what name others have given to the sounds we hear, and thus to avoid serious misunderstandings and confusions of nomenclature. Tubular and bronchial breathing, harsh breathing, rales and rhonchi and crepitations are illustrations of disputed points in affixing labels to aural perceptions. I here assume tubular as identical with bronchial breathing, and shall endeavour in the succeeding paragraphs to follow my own definitions of the remaining sounds.

Having thus cleared a good deal of the ground of symptoms and physical signs, we will now proceed to discuss some of the diseases on which the phenomena depend.

LARYNGEAL AFFECTIONS

The symptoms that are likely to call attention to the larynx as the seat of disease are :—

(1) Pain felt locally.
(2) Cough, at first almost certainly useless, and only later, if ever, useful, *i.e.* with equivalent expectoration.
(3) Especially a combination of (1) and (2).
(4) Alterations in voice, hoarseness, huskiness, aphonia, etc.

N.B.—In one great and dangerous group of laryngeal affections, viz. paralysis of abductors from disease at a distance, there are very often no symptoms whatever (if a possible slight shortness of breath be excepted), and the condition is only discovered by laryngoscopic examination, either as a routine matter or because other features of the patient's complaints have suggested that a paralysis might be found.

The causes to which those symptoms may be due are inflammation, acute and chronic, new growths, and paralysis of laryngeal muscles. To endeavour to associate symptoms with causes and to make an exact diagnosis without a laryngoscopic examination is simply to attempt the impossible, and in what follows the final necessity of such examination must always be assumed, though a strong degree of probability may certainly be arrived at by a fair consideration of the history and circumstances of the case.

Hoarseness of voice, if it occurs in a child, is almost certainly due to either acute laryngitis (simple or membranous) or to papillomata of the cords. Ulcers of syphilitic, tubercular, or malignant nature are practically unknown in childhood, and so is paralysis

of the cords, except post-diphtheric, and then the history is tolerably obvious on slight inquiry. The acute pyrexial illness with catarrh and obvious faucial congestion will easily decide that acute laryngitis is present, but only the laryngoscope will prove that there is or is not also a papillomatous condition, or show a case of pure and primary false membrane of the larynx. In adults the same hoarseness of voice may have the ulcers above mentioned as additional causes, but not every conjunction of apical phthisis with hoarseness, of gumma of the liver, with the same voice disturbance, has distinct ulceration of the cords as the necessary and sole cause of this latter symptom. Phthisical and syphilitic patients are not exempted from a simple laryngeal catarrh.

The following table gives the main points of distinction between the various forms of—

Ulcers of the Larynx

Tubercular.	Syphilitic.	Malignant.
Slow in progress, and not much destruction till a long interval has passed.	Rapid in extension when once broken down.	Intermediate in rapidity.
Shallow, superficial ulcers, without much swelling or infiltration.	Deep, ragged, sloughy ulcers, with much tumefaction.	Ulcer itself not very deep, but much excess of tissue formation, and not much simple swelling.
Curiously painful on swallowing; commoner from say twenty to forty-five years.	Little painful; commoner from say thirty-five to fifty.	Little painful; commoner over fifty.
Almost certainly evidence of pulmonary infection of tubercle, of chronic type.	Pulmonary association of a chronic type; very rare; may be septic aspiration-pneumonia.	If lungs affected at all it is with acute septic bronchopneumonia. (Schluckungs-pneumonie.)
Cords freely movable.	Cords not so free.	Cords often quite fixed and immovable.

Laryngeal Paralyses

The functions of the larynx with its vocal cords are three. These, with their natural nerves and muscles, may be thus tabulated:—

Function.	Action of Cords.	Muscles.	Nerve.
Speech	Approximated	Arytenoids, lateral crico-arytenoids	Recurrent laryngeal.
	Made tense with varying tension in voice expression	Crico-thyroid	Superior laryngeal.
Cough (or extraordinary respiration)	First opened by	Posterior[1] crico-arytenoid	Recurrent laryngeal.
	then firmly closed by	All the other muscles	
	then suddenly opened by	Force of air and relaxation of adductor muscles. Possibly by action of posterior crico-arytenoid	
Ordinary breathing	Very little movement, but what there is tends to allow free entrance, with gradual exit of air	Slight abduction in inspiration? Slight adduction in expiration? (No movement according to Semon)	Recurrent laryngeal.

We should naturally expect, then, that paralysis of muscles should interfere with some or all of these functions. In the main the inference is correct, but the exceptions are very important. The affection may be uni- or bi-lateral: it may be of adductors or abductors, or both. The results, with their reasons, are as follows:—

Unilateral Abductor Paralysis

Speech. Very little or not at all affected, because (1) the cords can still approximate; (2) they can still be made tense by the crico-thyroid.

[1] Even for a student fairly well up in anatomy it may be a little difficult to remember the action of the laryngeal muscles. The O in posterior may help him to remember that this is the only muscle which always and consistently Opens the larynx.

Cough, or extraordinary respiration.	In the later phases of the act unaffected; same reasons as for speech. In its first violent inspiration possibly a little difficulty or stridor, because extraordinary inspiration requires the larynx to be as widely open as possible.
Respiration.	Ordinary, unaffected, because the slight respiratory movement can easily be affected by the other cord.

Bilateral Abductor Paralysis

Speech.	Still frequently unaffected. Reasons under Unilateral Paralysis.
Cough.	In later stages of the act unaffected, because—reasons above. In violent inspiratory phase probably considerable difficulty or stridor, because the lax cords may be drawn together by the draught of air, and completely close or seriously narrow the aperture of entrance.
Respiration.	Ordinary, probably unaffected, but may be occasional severe dyspnœa. Reasons under Inspiration of Cough.

Unilateral Adductor Paralysis

Speech.	Probably unaffected, because the tensor is still sufficient to put the cord in a vibratory condition. Hence a further deduction may be made, that if the voice is altered, and only unilateral adductor paralysis seen, the probabilities are in favour of a source for the paralysis high up, before the superior laryngeal nerve is given off.
Cough.	In later explosive phase probably weakened, because the air begins to escape past the lax cord as soon as pressure increases in the expiratory effort. Extraordinary inspiration unaffected.
Respiration.	Unaffected to any appreciable degree, because air can get freely in, and the exit aperture is so little increased.

Bilateral Adductor Paralysis

Speech.	Even now may be unaffected materially except for shortness of breath through loss of expiratory control, because—*vide* argument above in unilateral trouble, which still holds good.

Cough.	Later explosive stage almost nugatory, because air now escapes very freely directly any excess of pressure behind is produced by expiration. Extraordinary inspiration unaffected; aperture of entrance very free.
Respiration.	Probably a little quickening of respiration, because of the loss of control over expiration, so that air escapes too freely.

Total Laryngeal Paralysis

Speech.	May even now be nearly perfect as regards tone, but shortness of breath will be noted in continuous speaking, because—if the tensors have escaped they are still sufficient, or nearly so, for tone in single words, but adductor paralysis will cause shortness of breath (*vide* above). If voice is quite lost, and tensors also paralysed, we are justified in diagnosing a central lesion in the medulla affecting both roots.
Cough.	Non-explosive and nugatory, because of adductor paralysis. Inspiratory phase, possibly dyspnœa and stridor, because of adductor paralysis.
Respiration.	Ordinary, as in all the others may still be unaffected for the reasons above.

N.B.—An opposite and complementary line of reasoning may be applied to spasms of the larynx, which are much rarer and only in laryngismus stridulus seen to perfection.

Into the actual causes of these paralyses I do not propose to go very deeply here; a few will be noted below under Tumours of the Thorax, and the subject will be apparent again in the nervous section. The following outline classification is, however, useful; it is mainly after Sir F. Semon (*B. M. J.*, January 1, 1898), but rearranged:—

Functional.	Toxæmic.	Lead, arsenic, and possibly other metals. Diphtheria, typhoid, rheumatism, etc.
	So-called hysterical.	

- Organic.
  - Primary disturbance in the central cells of medulla or cord.
    - Bulbar paralysis.
    - Tabes dorsalis.
    - Disseminated sclerosis.
    - Hæmorrhage.
    - Softening. Tumours of medulla.
  - Primary disturbance in the nerve trunk.
    - Such cases of the toxic group as have ended in permanent destruction.
    - Pressure of aneurysms, malignant tumours, inflammatory thickening (pleural, pericardial, meningeal).
    - Syphilitic processes similarly situated to inflammation.
  - Traumatism.
    - Surgical.
    - Suicidal.
    - Accidental.
    - Homicidal.

The point of greatest practical importance is to determine whether a given paralysis is organic in origin, with practically no chance, or functional, with a fair chance, of recovery. For the toxæmic group of functional cases the great point is to be aware of their existence, and should paralysis be discovered a history of previous or coexistent illness must be carefully inquired for, and its possible treatment by metallic drugs investigated. The affection itself has no distinguishing features, though it may be expected to be of a bilateral type owing to its origin in circulating toxins.

For the so-called hysterical paralyses the following points are probably sufficient :—

1. The patient will come with a predominant complaint of loss of voice.

2. Close physical examination will reveal no gross organic lesion, but will probably show—

3. Other features of nervous instability.
4. The sex will probably be female.
5. The age will usually be under thirty.
6. The type of paralysis will be adductor and tensor, the combination of the two indicating a cerebral origin, which this undoubtedly owns.

As no danger (of asphyxia at least) can arise from adductor paralysis, *vide* table for reasons, the mental association of this form with the usually good prognosis in hysteria may help the student to remember the clinical fact that by far the majority of cases of adductor paralysis are functional, while a much greater proportion—in fact, all cases of abductor paralysis—are of grave and probably very serious import.

PNEUMOTHORAX

Is a condition in which air in the sac separates the two layers of the pleura; this may occur either over the greater part or whole of the lung, or over much smaller areas limited by previous adhesions. It is a condition easily liable to be overlooked or misunderstood, and yet of frequent occurrence, so that it is well we should understand the principles of its natural history and mechanism, which are not, I think, sufficiently clearly set forth in most text-books.

In origin, then, the gas must be :—

1. Admitted from without.	Traumatism.	
	Surgical procedures.	
	External fistula opening into sac (rare).	
2. Developed *in situ*.	From decomposition of a pleural effusion (also rare).	
3. Admitted from within.		
(*a*) Bursting of a gas-holding viscus or cavity other than pulmonary.	Œsophagus (cancer, etc.)	All rare.
	Stomach (ulcer, cancer, etc.)	
	Subdiaphragmatic abscess.	
(*b*) Laceration of visceral pleura and lung.	Phthisis (most commonly).	
	Abscess or gangrene.	
	Emphysema.	
	Violent respiratory efforts in healthy lungs (very rare).	
	Penetration of broken bone through pleura.	

Admitting that the condition exists, the diagnosis of the source, according to the above table, presents but few difficulties. In the first group, "Admitted from without," both the cause and the condition are obvious, and require no discussion. In the second group, "Developed *in situ*," the pleural effusion will probably have already attracted attention; the smell of a little fluid withdrawn by aspiration will leave no doubt about decomposition: such cases are, however, extremely rare, and our further analysis will be partly applicable to them. In group 3 (*a*) the previous symptoms will probably have made the source clear if the pneumothorax be discovered, as, indeed, they will also probably have done in group 3 (*b*), but it is especially to this last group that our argumentative analysis applies for the estimation of the occurrence of the accident.

We must first claim one or two postulates which are, I think, justifiable:—

Postulate 1. That the pleura must be healthy (or at least not adherent). Over the area of separation this must be true, except under conditions of gaseous pressure too high to take into clinical consideration.

Postulate 2. If the pleura be healthy or non-adherent, the admission of air to its cavity must allow of some collapse of the underlying portion of the lung.

Postulate 3. This collapse must diminish (it may be in slight degree) the utility of the affected lung.

Postulate 4. Laceration of healthy pleura must be attended by some pain if consciousness is still present.

Postulate 5. (Not quite so obvious, but justified, I believe, by post-mortem experience.) The opening into the pleura must be (*a*) a round hole; or (*b*) a valvular rent; or (*c*) a slit more or less irregular.

Postulate 6. That the admission to the healthy pleura of air or any foreign material will cause cough of greater or less severity.

Granted these postulates, we may divide the immediate symptoms of the occurrence of pneumothorax into the inevitable or constant, and the quasi-accidental or variable.

The Inevitable or Constant are:—

1. Cough.—This is, in fact, the actual cause of the accident, by the increased air pressure it produces in the lung; so while usually preceding pneumothorax it will certainly continue after it, except in cases of sudden death.

2. Pain in the side.—Must be present by postulate 4. It is usually sufficient to attract attention, but may be so slight or so mixed up with other discomfort as only to be found by leading questions.
3. Shortness of breath (*vide* under Quasi-Accidental).

The Quasi-Accidental or Variable Symptoms are really only one, viz. shortness of breath or dyspnœa (the quickened pulse, the collapse, the sweating, etc., are merely variable concomitants of the amount of asphyxia). This shortness of breath may be so slight as to be quite unnoticed, or may be so severe as to lead to death, appalling in its suddenness, with every conceivable degree between the two. For the primary explanation of the degree we must draw a distinction between the symptoms setting in immediately with the accident, and those which develop in the course of a little time. For that which comes on at once, one quite sufficient explanation is obvious, viz. the share in aeration of the blood previously taken by that portion of the lung now collapsed and useless. That which arises in continuance of the primary trouble may own this same explanation, but probably in association with another, viz. the possible valve-like action of the torn pleura allowing an inspiratory pumping-in of air to the sac, but preventing expiratory escape, so that more and more healthy lung is allowed or forced to collapse; this may go on to a degree incompatible with life, or by the establishment of pressure and tension equilibrium it may remain stationary, life being still possible. In all cases and under all circumstances the ultimate degree of shortness of breath has then the above foundation, viz. *the previous utility of the collapsed area.*

The physical signs of pneumothorax, *per se* and apart from the disease causing it, may be naturally divided into (*a*) those found over the area of separation; (*b*) those observed elsewhere.

(*a*) The former will be:—

On inspection.—Nil, unless a large area of lung be collapsed, when that side of the chest may be a little sunken, and probably deficient in movement.

On palpation.—Nil, unless the opening into the sac be a free one, when increase of tactile vocal fremitus may be appreciated, owing to the freer conduction of aerial vibrations with a resonating chamber.

On percussion.—Hyper-resonance, owing to the air coming into immediate contact with the chest wall, and so allowing unusual vibratory opportunities.

On auscultation.—The signs will vary from tubular breathing with amphoric resonance—if the opening be patent—to complete silence when the lung is collapsed, and the opening a mere slit, so that the air pressure in the pleura prevents the lung from expanding.

(*b*) The latter refer to (1) the increased work thrown on the still functionating area of lung, leading to increased loudness of the breath sounds previously heard, of whatever nature; (2) the displacement of the heart's apex beat, which may be much or little, right or left, according to the amount and side of the collapsed area of lung; (3) frequency of pulse and severity of asphyxial symptoms, dependent as before on the previous utility of the portion of lung now *hors de combat*, and possibly in some degree on the cardiac dislocation.

The sequelæ and subsequent history of a case of pneumothorax depend upon the admission to a living serous membrane of foreign material. If this is merely aseptic air, and the opening is such as can readily be healed, recovery is likely to be complete with the restoration of the *status quo ante*. If it be tubercular or septic, it is almost inevitable that pleurisy of a corresponding type shall arise, with its appropriate symptoms.

With this analysis, the diagnosis of pneumothorax can hardly require further discussion, bearing in mind that it always occurs suddenly as an acute episode in health or chronic disease. It is, however, somewhat frequently assumed that the condition may be mistaken for a pathological cavity in the lung. When, as is frequently the case, the two coexist, diagnosis is difficult, I admit, and probably immaterial; but under other circumstances the two offer more contrasts than likenesses, as may be seen by comparing the above with the following:—

INDICATIONS OF A PULMONARY VOMICA

On inspection.	Sinking in of chest from the old fibroid disease which remains from the original trouble that caused the excavation.
On palpation.	Increased T.V.F. from the consolidation surrounding the cavity, and from the resonance of the chamber.
On percussion.	Dulness and increased resistance, again arising from induration and consolidation around the cavity.
On auscultation.	Tubular breathing with amphoric resonance, frequently adventitious sounds of the nature of rales, and clicky or resonating crepitations; the former arising in and from the cavity, the latter from the fluid in it and from the consolidating catarrhal mischief going on around it, so that their absence is a good sign as indicating inactivity of disease or its obsolescence.

These are the signs usually ascribed to a vomica in the lung, and the reasoning is sound as far as it goes, because chronic phthisical processes (tubercular or non-tubercular, *vide* below, Fibroid Phthisis) are far and away the commonest causes of these excavations. But even with this causation there are two or three conditions that must be fulfilled before the signs will be present in typical form: (1) it must be fairly superficial to prevent its appropriate signs and sounds being overwhelmed by those of overlying healthy or diseased lung; (2) it must be of fair size, at least as large as, say, a nut, to cause amphoric echo; and (3) it must communicate by a patent opening with a bronchus to allow access of air. The two acute processes, viz. abscess and gangrene, which may produce rapid excavation of the lung, are by their very acuteness sufficiently distinguished, *qua* excavation, from tubercular mischief.

N.B.—It is advisable, or even imperative, in doubtful cases of excavation, to listen to the chest actually *while the patient is coughing*, as well as to note alterations in signs after a cough, because the tube or tubes leading to the cavity may be blocked with material only to be removed by coughing.

PHTHISIS

"An assemblage and progression of symptoms associated with and dependent upon the ulcerative, or suppurative, destruction of a

more or less circumscribed non-malignant deposit in the lungs," was the late Sir Andrew Clark's definition of the disease, and I do not think a better one could be devised, although it excludes miliary tuberculosis, for it is wide enough to embrace all the non-tubercular chronic destructions of the lung which are associated anatomically with suppuration and ulceration. Both before and since the enunciation of this definition an enormous amount of literature has appeared containing exhaustively detailed descriptions of the anatomical results of phthisis in the lung, and classifications of the disease of such endless variety, that the student of the subject must get confused in trying to get an intelligent idea of the whole of it; for under such a weight of exposition he is in great danger of losing sight of that unity of principle with diversity of result which underlies and explains the varying aspects of phthisis and chronic diseases of the lungs. It is to this unity of principle that I wish to draw special attention, with a few brief notes on the results and conclusions.

The frequency of tubercular—as compared with all other forms of—chronic irritation and destruction of the lung, and the special anatomical results of this special form, have led to a tendency to view inflammation in the lung as always something special, with special laws of its own. Such a view is erroneous and misleading, and the first great principle in our present subject is that the lungs offer no exception to the rules that govern the processes of so-called inflammation, as it occurs amongst vascular tissues anywhere in the body. These processes we must briefly indicate in a series of paragraphs:—

1. The collective phenomena known by the name of inflammation are the invariable result of a locally acting (not necessarily locally produced) irritant *of any kind whatever*, provided this be not sufficiently severe to kill the tissues outright; and then, be it remembered, the processes go on in the surrounding living tissue, the dead mass sharing in, or being the efficient cause of, the irritation.

2. The first phenomenon is interference with the freedom of circulation through the part, a coincident out-soaking of fluid, and the appearance in the area of numbers of small round cells. The majority of these cells are of feeble vitality, if not actually dead, representing as they do to a large extent the dead and wounded in the fight between invaders and defenders. The tissues, too, in and near which the fight is going on, suffer in vitality from the disturbance in their normal circulatory refreshment; this is the *functio*

læsa of inflammation, whether in mesoblastic or epiblastic elements.

3. Paragraph 2 represents the destructive phase of inflammation. It may reach any degree of intensity, from the microscopic-in-quantity-but-identical-in-quality of the healing of an aseptic wound up to the quarts of pus and sloughing of septic cellulitis; and it continues in the immediate neighbourhood of the irritant so long as this—or its products—remains in such a condition and position as to come in contact with irritable tissues.

4. Following this, as the defenders locally or generally get the upper hand, comes the reparative phase in which granulation tissue is formed, consisting of new nucleated cells (the precise origin of these and of pus, though perhaps important, has no bearing on our present purpose), endowed with vitality because of, and in proportion to, their proximity to supplies of nutriment in the shape of newly-formed blood capillaries and intercellular lymph spaces.

5. Following granulation tissue comes the formation of fibrous or scar tissue, which possesses primarily some peculiar properties: (*a*) it is practically non-irritable, hence should the irritant still remain active *in situ*, it serves to shut it off from its field of operation on irritable tissues, and eventually causes its death by isolation; (*b*) though poor in vitality itself, it is not irritating to healthy tissues, and has no tendency like dead material to cause a spread of inflammation; (*c*) it is extensile and contractile, almost mechanically so, so to speak, very different in these qualities from naturally existing fibrous tissue, exhibiting either quality to an extreme degree, unbalanced by intermingled muscular fibres; (*d*) though in general it is produced in an amount which is simply reparative, it is occasionally liable to excessive growth, as is seen in false keloid and in some thickened pleura.

6. The dead material that arises as the result of the destructive phases of inflammation, if left *in situ* and isolated by scar formation, and if it does not become the seat of a fresh development of irritating energy (septic microbes, etc.), has a tendency to dry up and be converted into cheesy material, and ultimately into a calcareous mass by the deposition of lime salts.

7. The two phases of inflammation—the destructive and the constructive—must go on so long as the irritant remains in contact with irritable tissues. The fight must always be to the death or impotency of the tissues, or of the irritant. Hence in the case of a living and propagating irritant (microbes) it may be prolonged indefinitely; the microbes striving, and, alas! too often with over-

whelming success, to cause destruction, the tissues as continuously, but with indifferent success, to efface their enemies by scar formation.

8. From a given primary focus inflammation may spread by (*a*) direct continuity as the irritant extends its area of influence wider and wider (this method of extension, if of any distinct degree, is almost conclusive proof of microbic invasion, for a simple mechanical irritant cannot spread; a non-reproductive chemical one would rapidly become neutralised by combination; and it is only by direct multiplication of the microbe, or by the continued production of toxins, toxicogenic zymins, etc., that we can understand the widening area of irritation); (*b*) mechanical transference of the irritant to other points, either by the blood stream or per lymphatic channels, or, especially in the lungs, by air currents or mere mechanical dropping under the influence of gravity into parts which are for the time being lower than the original focus.

These are the essential principles of inflammation wherever occurring, undeviating in their action, only modified, not obscured or fundamentally altered, by the nature of the invading irritant and the elementary anatomical details of the organ attacked.

Of the modifications introduced by the nature of the irritant, we must note that in tubercle (the same is true to a certain extent of the other infective granulomata) there is a special tendency to the early and rapid formation of a small mass or nodule of granulations with a vitality and cohesion somewhat in excess of that met with in the results of the inoculation of more virulent microbes. These nodules would apparently seem to belong to the reparative side of inflammation, for they do occasionally—it is true very rarely—become quite fibrous even in a miliary tubercle, but owing to their low degree of vitality and lack of capillary nutrition they cannot individually grow to any size without caseating or softening down. Owing to the great pertinacity and multiplication of the irritant they are, however, persistently produced in the immediate vicinity of the original focus, and by their union and coalescence may constitute masses of caseous material as large as the thumb or larger. If the irritant be of a more virulent type, the destructive phases of inflammation are more prominent, with suppuration and tissue death (microscopic = necrosis, macroscopic = gangrene), and, possibly, the development of foul smelling gases as the predominating processes. If different irritants of different virulencies be at work at the same or successive times, the results are likely to vary according to the above sketch, first one and then another becoming the predominat-

ing feature of the case, *e.g.* catarrh followed by tubercle, pneumonia by gangrene.

Of the modifications introduced by the anatomical peculiarities of the lung, we have only to note the air passages with their enormous area of mucous membrane, lined with epithelium, to which air must necessarily be freely admitted; air, too, which cannot always be filtered through nasal vibrissæ, and which consequently often, or even usually, contains multitudes of microbic, mechanical, and chemical irritants of every kind. Hence we have (1) an extraordinarily extended frontier offering opportunities of invasion; (2) the most favourable opportunities for the accumulation, in tubes through which air currents are feeble, of mucus and dead epithelial, and other cells (catarrhal products), on which microbes may implant themselves without opposition, and grow and multiply till they reach irritable tissues; (3) the *physical signs* of such tubercular nodules or of catarrhal and pneumonic products will naturally be essentially identical in local details over small areas, for they all produce consolidation or collapse of lung tissue, or sticky fluid contents for the air tubes; it is this which causes so many difficulties in diagnosis.

We may now apply these principles and their modifications to explain the correlated pathology of, and the adjectives applied to, the varieties of phthisis.

Pneumonic Phthisis

This term may be used to refer either to the amount of consolidation of the lung, or to the clinical history of the case, resembling at some period of its course a pneumonia (croupous or catarrhal). In the former case we have the tubercular nodules rapidly forming and coalescing (scrofulous or acute pneumonic phthisis of authors) until large areas are involved, giving the physical signs of a pneumonia, but without the definite limited symptoms of that trouble; the softening of the masses will add still further likeness to the resolution stages of ordinary pneumonia. For the latter signification of the term, *i.e.* a clinical history resembling that of a pneumonia, it is a common enough event for the tubercular nodule to cause an inflammation of a non-tubercular nature to surround the original focus with consolidation (tuberculo-pneumonic phthisis of authors), and this may occur at any period of the progressive destruction. In both these cases the tubercular bacilli are the original irritating invaders. But another common sequence is the primary invasion of the pneumococcus (croupous) or of less definite microbes (catarrhal), causing a wide-spread con-

solidation or pneumonia, and in the weakened tissues and dead material thus produced the specific tubercle bacilli find a favourable nidus for growth, and proceed more or less rapidly to continue the mischief after the acute symptoms of the original invasion have subsided. This sequence of events explains the term catarrhal phthisis, though the *term* might equally well be applied to the ordinary cases of not very acute tuberculosis of the lung in which large areas are not simultaneously solidified.

Fibroid Phthisis

This term includes *all* varieties of chronic lung destruction, of whatever nature, in which the reparative or scarring phases of inflammatory reaction have reached a considerable degree and extent. Inasmuch as the bacillary or simple irritant invasion is entirely a matter of accident, we have the following easily understood varieties:—

Tubercular. Another name or way of expressing a very chronic case of tubercular phthisis, in which the fight being a prolonged one, the scarring is very evident. (The term must obviously be also applicable to cases of cured phthisis.)

Tuberculo-fibroid. Nearly the equivalent of the first variety, only that subsequent to the tubercular scarring other irritants continue the inflammatory reaction and healing.

Fibro-tubercular. In which the primary scarring is due to indefinite bacillary or even mechanical (pneumonokoniosis, stonemasons' or coalminers', etc., lung) irritants, and on the weakened tissues the tubercle bacillus makes a descent, and continues the destruction-with-healing processes.

Fibroid. A condition of fibrosis of the lung, starting as above (most commonly, perhaps, from an unresolved pneumonia or bronchopneumonia), but in the production of which the tubercle bacillus never at any time plays any part.

N.B.—Post-mortem experience would almost lead me to believe that in any of these cases the scar may take on a keloid-like extension, independent of obvious irritation.

Hæmorrhagic Phthisis

When hæmorrhage of noticeable extent occurs as the result of "ulcerative destruction of the lungs," the blood may either coagu-

late *in situ* round the focus, or it may also be mechanically carried into distant air tubes and alveoli. The ulceration may or may not be due to the action of tubercle bacilli; in the former case, extravasated blood is likely to be more or less impregnated with the bacilli, and in proportion as it is impregnated it acts locally or at a distance, like a laboratory plate culture, causing a rapid multiplication of the points of infection. (This is the old phthisis *ab hæmoptoe*. The older pathologists did not recognise, as we now do, that the bacilli were there before the hæmorrhage, and that it is only their extension which is due to the bleeding.) In the latter case, when tubercle bacilli are not present, the hæmorrhage, *qua* hæmorrhage, is harmless enough, or, at least, has to be considered from a totally different standpoint of causation.

Miliary Tubercle

Is a condition in which thousands of tiny nodules are scattered through the lungs (and other organs, liver, spleen, etc., including tubercular meningitis), and only requires notice here to insist upon the fact that it is *never a primary form of attack from without of the tubercle bacillus*. It invariably arises from some caseous or softening tubercular[1] focus within the body, the bacilli being carried thence by lymph or blood stream. This focus can always be found by a sufficiently close search on the post-mortem table.

Diagnosis of Pulmonary Tuberculosis

From the above consideration of the pathology of, and anatomical changes in, chronic and even acute pulmonary disease, it will be seen that an accurate and exact diagnosis of their *causa causans* is not to be obtained by physical signs alone. All such affections are, or may be, indifferently associated with crepitations, rales, and rhonchi, and with consolidation of small or large areas of lung, with or without signs of pleuritic involvement. We may be inclined in one direction by the localisation of these morbid phenomena to an apex or to a base, and led in another by their universal distribution over the whole of one or both lungs. The

[1] In all that relates to phthisis the terms "tubercle" and "tubercular" are used in strictly concomitant association with assumed specific bacilli. The terms "nodule" and "nodular" are used in their strict meaning of a little lump, or referring to a little lump. It is the confusion in these terms that has led to much confusion in translating the meaning of the older authors, who made tubercle the equivalent of a little lump, without reference to the presence or absence of bacilli, of which, indeed, they naturally knew nothing.

family history may show a special proclivity to tubercular disease.[1] We must weigh the circumstances of occupation as affording opportunities for pneumonokoniosis; we must note with anxiety a failing appetite, with wasting and sweating, and carefully watch the progress of our patient when acute symptoms (cough, fever, hæmoptysis, etc.) have subsided. But when all these points have been duly weighed in the balance, remembering that tubercle may precede, accompany, or succeed any other irritant, we shall still have before us the one crucial question, "Is the bacillus tuberculosis present in the expectoration or not?" Universal consensus of opinion warrants the statement that if the bacillus be found, it is at any rate a prominent sharer, if not the sole factor, in the production of the patient's illness, so that if we would guard against disappointment in prognosis and errors in diagnosis, the search for the microbe must be undertaken in every case permitting of doubt. The most likely cases for error and doubt are the following:—

(a) *Bronchitis and Bronchopneumonia in Babies and Young Children.*—Here sputum for examination is practically unobtainable, and we must rely mainly on the following considerations:—If the disease is simple in origin the patient should get well in from two to three weeks, and the improvement in the child should correspond fairly well to the clearing up of the morbid lung sounds. If the child does not rapidly pick up after this time, a very careful search must be made for empyema, which very commonly develops and retards convalescence. Should no evidence of pleuritic trouble be found, and the child still not improve, then tubercle must come strongly under suspicion, and every system and part of the body must be carefully examined for corroborating or contradicting evidence. Pulmonary tubercle under the age of say ten or eleven years is practically only of one kind, viz. miliary, or at least very widespread, and hence a wide area of persistent morbid phenomena will be suggestive. Those other unknown irritants which maintain a chronic catarrhal condition have a contrary tendency, viz. one to preserve their morbid activity only in one locality, and that usually at the base. These basic chronic catarrhs, especially if occurring after a specific fever, such as measles, by no means infrequently persist, and form the origin of a genuine purely non-tubercular fibroid phthisis.

[1] Modern opinion, in more strict accordance with the bacillary or infective theories of tubercular processes, is inclined to attribute more weight to the view of direct infection by personal contact with a sick relative or friend, or by inhalation of germ-infected air of the sick-room, and less to the mere possession of tubercular ancestors.

(b) *Repeated attacks of "Cold and Cough," with slight symptoms of Mild Bronchitis.*—This group of symptoms constitutes the very commonest history of the commencement of an ordinary case of phthisis, and must, therefore, *ab initio*, arouse our strongest suspicions. Sputum may be available, and may decide the diagnosis out of hand, but it must be remembered that even tubercle takes a little time to soften and ulcerate, so that expectoration may be unobtainable, or, again, its examination may yield merely negative results. We must then proceed to carefully examine the lungs for a small localised area over which the auscultatory phenomena are not only pathological, but different from the universally heard rhonchus of bronchitis, and suggestive of consolidation. Such are commonly, but by no means invariably, found at the apices of the chest. They are found frequently enough at the apex of a lobe, or, in my experience, often in the neighbourhood of the pericardium. I do not think that a visible want of expansion or altered percussion note is of much use in deciding doubtful cases. By the time they are definite enough for observation the general symptoms have rendered their aid superfluous. I am accustomed to rely more upon the sharpness of the crepitations—"clicky" is the term I usually apply to them—and a slight increase in voice and *expiratory* breath sounds. If these go with a pulse frequency, and a loss of weight and appetite out of proportion to the apparent illness, I feel at any rate judgment must be suspended, and the case declared one of doubtful and suspicious nature, and the more suspicious, the stronger the lead given by the patient's history, whether family or personal. These are the cases that made our predecessors in medicine divide their patients up into constitutional types of liability to consumption: the blonde, with thin flaxen hair, the brunette, with flushed cheeks, the sprightly or ethereal, the clumsy or scrofulous; their descriptions are still true in the main, for they were founded on an experience which is still ours.

(c) *Hæmoptysis.*—It may be said at once that genuine pulmonary hæmoptysis, and especially if free or repeated, when found with the physical signs and symptoms considered under (*b*), adds so enormously to the probabilities of tubercle as to render diagnosis nearly certain; but the symptom is so common and so alarming to our patients, that it may be discussed at a little greater length. We have already (p. 54) enumerated and briefly mentioned the possible causes of hæmorrhage, and on p. 152 given the diagnosis between hæmoptysis and hæmatemesis. It remains here to consider the question commonly enough asked, "What do you expect to hear

in the chest of a patient who comes to you complaining of 'spitting of blood?'" We may, I think, reply accurately enough by saying, "If blood-spitting is his *only* complaint, it is probable that we shall hear nothing." The reasons for this reply are: (1) that in a very large proportion of such cases the blood comes from elsewhere than the lungs—gums, pharynx, nose, larynx, etc.; (2) that in again a large proportion of the cases in which the blood does really come from the lungs, it is symptomatic of *advanced* heart or lung disease, which will have given rise to marked symptoms previous to, and different from, the hæmorrhage; and (3) it is a common experience that we hear nothing—not even the crepitations of blood in the bronchioles—in those cases which time shows only too clearly were really cases of tubercular hæmorrhage. In connection with the diagnosis of the causes of pulmonary hæmorrhage, I cannot conclude better than by quoting a few sentences written by Sir Thomas Watson many years ago, and which still hold good. He says (*Lectures on the Principles and Practice of Physics*, 5th edition, 1871): "*Cæteris paribus*, the *disposition* to pulmonary hæmorrhage is increased by whatever tends to diminish the capacity of the thorax, and to compress the lungs, or the heart and great vessels. It is partly on this principle that we may account for the frequency of hæmoptysis in persons with crooked spines: in tailors who sit continually in a stooping posture; in young women who lace their stays too tightly; and even in those who labour under dropsy or other cause of distension of the belly." He then quotes in full detail a case of vicarious menstrual hæmoptysis, and proceeds: "It is, however, a melancholy truth that capillary hæmorrhage from the mucous membrane of the air passages is dependent in a very large proportion of cases upon incurable disease—tubercular phthisis or organic disease of the heart. When hæmoptysis is thus actually symptomatic of tubercular disease of the lungs, it is liable to considerable variety in regard to the period of its first occurrence. There are many persons in whom the first attack of hæmoptysis precedes, even for years, the primary symptoms of unequivocal phthisis. There are others in whom the first attack of hæmoptysis is immediately followed by all the signs which announce the presence of tubercles in the lungs." He then mentions heart disease and pulmonary apoplexy, and after quoting Andral to say that: "Of those individuals whom he had known to spit blood at some period or other of their lives, there was only one in five whom he did not *know* to have tubercular phthisis." He deplores his inability to alter the opinion he had long held and expressed: "That

if from any given number of persons who have been known to spit blood—excluding the *streaks* of bronchitis, the rust-coloured expectoration of pneumonia, and injury to the chest—we subtract those in whom that symptom was connected with irregularity in the uterine functions, and those in whom it was symptomatic of cardiac disease, there will be very few indeed left in whose lungs the existence of tubercle or other fatal disease may not be confidently predicated."

Except that we do not now regard pulmonary tuberculosis with quite such a hopeless feeling as regards prognosis, these statements of Sir Thomas Watson are, I think, as true now as when he wrote them.

(d) *Pneumonia that does not clear up rapidly after the Temperature has fallen.*—Here the very fact that the lungs do not clear up in, say, three or four weeks, constitutes in itself proof that *something* other than the pneumococcus is at work. This something may be tubercle bacilli or others not yet sufficiently identified (there is just a possibility that the lung tissue—the soil—of the individual may be peculiarly weak, and allow well known microbes to continue the irritation). Expectoration is always present, and can be obtained for bacteriological examination, so that diagnosis requires no further remark beyond calling attention to the fact that *an unresolved pneumonia is not necessarily at any time tubercular, but may at any time become so, and is, therefore, a standing menace to its possessor.*

(e) *Chronic Bronchitis.*—*Vide* below, p. 98. Here I can only say sputum is again available, and must be examined.

Of pyrexia in these various cases I have said but little, as its indications are of dubious significance. On the one hand, it is certain that a slight rise of temperature (101 or less) in the evening (especially with early morning sweating) is strongly suggestive of tubercle in those cases, as in group (*b*), where the physical signs are doubtful; but, on the other hand, it is equally certain that typical hectic temperature (102, 103, or 104 at night; 97 to 99 in the morning) is due more to septic processes, either with or without tubercle, than to tubercle itself, so that in the other groups we get very little assistance from thermometric observations.

There is only one other point in phthisis that I propose to mention, that is diarrhœa. It is important to remember that it may be due to any of the following:—

(*a*) Simple dietetic errors, from which phthisis is no protection. Inquiry into food will possibly suggest this cause.

(*b*) Simple irritability of alimentary tract, which is very common

in these patients, and may be increased by swallowing septic or irritating matter which ought to be expectorated. The diagnosis of this form can only be made by exclusion, and by noting that it moderates or ceases under appropriate treatment, and warning against swallowing sputum.

(*c*) Tubercular ulceration of gut. The diagnosis will mainly rest on the intractability of the diarrhœa, and possibly some tenderness of abdomen, together with an exclusive consideration of the other three conditions.

(*d*) Amyloid or lardaceous disease. This generally admits of easy diagnosis from the enlargement of the liver and spleen, and considerable quantities of albumen (without pus) in the urine. It is very rare for the intestine to be the sole or even the principal seat of the deposit. Should this, however, happen to be the case, it may be difficult or impossible to differentiate the condition from tubercular ulceration. In both cases the patient will probably be anæmic and cachectic; in both the flux will be persistent and intractable. We may remember that amyloid degeneration never occurs in early phthisis before suppuration has been long established; intestinal ulceration may, on the other hand, be a much earlier feature. Diarrhœa from amyloid disease, again, is usually stated to be entirely free from abdominal pain, while ulceration is frequently associated with colicky disturbances.

PNEUMONIA

Of the diagnosis of a well-developed attack but little needs to be said. The flushed or bluish face, with sweat standing in large drops on the forehead, the working of the alæ nasi, the rapid breathing, the painful efforts at subduing a distressing cough, makes up such a typical picture that we can often diagnose the disease at sight; and when woodeny dulness on percussion and tubular breathing on auscultation are added, the diagnosis is complete. But there are one or two points still that are worth a little discussion, even in an elementary work.

Diagnosis in Early Stages.—Primary or idiopathic pneumonia is particularly characterised by the very sudden rise in the temperature (probably 103 or 104 within twelve hours), and a corresponding sudden onset of symptoms; while the physical signs, being much slower in development, are still in abeyance. These symptoms are usually a rigor, or convulsions (convulsions in children often take the position of rigors in the adult as an indication of severe invasion

by microbes), speedily followed by headache with intolerance of light, frequently enough delirium, and vomiting of a nervous type, *i.e.* independent of food. Now these are precisely the symptoms suggestive of an acute toxæmia, or of intracranial inflammation; and hence many cases of incipient pneumonia may hastily be assumed to be something more dangerous than ultimately proves to be the case.

Careful attention to the following points may assist us in forming an opinion, but for twenty-four or even forty-eight hours it is often absolutely impossible to make a final diagnosis.

Inquire of the patient or friends as to any possible local source of infection, such as a wound, a boil, or an abscess, or even a blow on the head, and especially as to any discharge from an ear; the throat and ears must then be examined for signs of septic mischief, and time would not be wasted by asking if the patient has had the opportunity of acquiring an infectious disease from a sick friend. If negative replies are received to these inquiries, note the pulse-respiration-temperature ratio, *vide* p. 35, which even from the commencement is very likely to be markedly deranged in a case of pneumonia. Should the indications be still undecided, judgment *must* be withheld temporarily, until a cough with expectoration or physical signs come to our aid.

Central Pneumonia.—It occasionally happens that a pneumonia develops in, and remains confined to, the central part of one base, and thus the physical signs, even in the later stages, are concealed or ill developed. In such cases the sputum becomes a most valuable sign, and quite possibly gives an absolutely decisive indication; it is probably extremely sticky, but little aerated and non-purulent. If with these features it is *uniformly* stained with blood pigment, we are justified in saying that *some* pneumonia is present—the staining may be of any degree from rust to prune-juice colour, and the deeper it is, the worse the outlook.

Is Pneumonia a Specific Disease?—The facts suggesting such a theory are:—

(1) The sudden onset of symptoms before, and independently of, the lung consolidation.

(2) The sudden cessation of the symptoms while the consolidation still remains.

(3) The definiteness of the phases through which a typical case of the disease passes.

(4) The undisputed occurrence of small epidemics, and the appearance of attacks in which the evidence of infection from a

previous case is overwhelmingly strong (noticed in anxious wives or mothers too closely attending their sick charges).

(5) A microbe of very definite character has been pretty constantly found in sputum and lungs of typical cases; it has been isolated and cultivated, and to a limited extent has been put through the tests on p. 26.

Nos. (1) and (2) undoubtedly prove to a demonstration that the symptoms of pneumonia are not due to the lung consolidation as such, while (5) offers a plausible explanation of them; and we may, I think, sum the matter up by asserting that "One particular microbe is capable of producing a form of disease to which the term 'idiopathic pneumonia' is applicable, but that facts do not prove that this is the *only* microbe capable of producing a set of clinical phenomena indistinguishable from such pneumonias." Accurate post-mortem evidence, coupled with scrupulously exact bacteriological work, would, I suppose, clear up every case and prove its precise causation, but there can be no disputing the fact that consolidations of large areas of the lung, coupled with symptoms indistinguishable *per se* (*i.e.* apart from the clinical history of the case previous to their onset) from those of an ordinary pneumonia, do occur in the course of bronchial catarrh, of puerperal, and other septic troubles, and of influenza amongst many other diseases. Admitting this, it is difficult to believe that the lung mischief is necessarily due to diplococcus pneumoniæ, and that we must deny to other pathogenic organisms—specific or common, known and unknown—this power over the lung. Again, on the other side of the question, the diplococcus pneumoniæ has been found frequently in morbid effusions in joints and elsewhere, under circumstances strongly suspicious of a causative relation, so that it cannot be said to confine its attention to the lungs.

The only possible deduction from these facts is to give to clinical pneumonia a modified specificity, perhaps equal to that given to clinical diphtheria (*q.v.*), while denying to it one so complete and unequivocal as that given to measles, syphilis, or scarlet fever.

ACUTE BRONCHITIS AND BRONCHOPNEUMONIA

The late Dr. Sutton used to teach that healthy children did not suffer from bronchitis. If this be so, then the zymotics, poor feeding, and neglect are responsible for much subsequent disease amongst our young population, for bronchitis is a very common

disease in infants, and as bronchopneumonia is a direct extension of the morbid processes to the bronchioles and alveoli, it is a common difficulty to decide whether a given case shall be called bronchitis or bronchopneumonia. My own opinion is that practically all cases of bronchitis amongst young children, if severe, have some degree of alveolar extension, so that from the point of view of treatment the diagnosis is of little moment, being more of academic or examinational interest. If dulness on percussion and tubular breathing are present over localised areas they settle the matter at once, but as these are easily obscured by non-affected lung tissue and the rhonchi of the bronchitis, and as children are awkward at, or incapable of, deep voluntary respiration, I usually rely upon the following two features: (1) *The Temperature:* if 101° or over, it points to alveolar extension; if below 101° and yet above normal, it points to uncomplicated bronchitis; if markedly subnormal, it again points to very dangerous and universal bronchiolar involvement. (2) *Rales and crepitations:* if these are so abundant as to markedly predominate over the rhonchi of bronchitis, I again assume that bronchopneumonia is present. Dr. Osler, in his deservedly popular work on medicine, will not allow any difference between capillary bronchitis and bronchopneumonia. As regards the *nature* of the morbid process he is obviously right, for one cannot draw an imaginary mathematical line between bronchioles and alveoli; but as regards *extent in area* I think he is wrong, and I would define bronchopneumonia as a local (few or many patches), capillary bronchitis as a universal, extension of the catarrhal process to fine tubes and alveoli. This definition best explains the undoubted difference in prognosis; capillary bronchitis I regard as uniformly and inevitably fatal, while recoveries from local consolidation are numerous and, indeed, the rule.

CHRONIC BRONCHITIS AND ITS ASSOCIATIONS

It is a very common observation that one attack of bronchitis (irrespective of the nature of the irritant causing it) predisposes the sufferer to another. A little consideration of the pathology of inflammation easily explains this. It is almost inevitable that, in a mucous membrane that has once been inflamed and shed a good deal of epithelium, slight scar changes, diminishing its vital resistance, should be left behind. Proceeding further, if we allow that these changes (scar tissue) will diminish the natural vital

elasticity of the tubes, it is easy to see that with repeated attacks two consequences are likely to follow: (1) the stiffening process will extend farther and farther down the tubes, or become more and more marked in local segments of them; from this it will follow (2) that increased air pressure, whether expiratory or inspiratory matters not, must be felt more and more by the smaller air tubes and alveoli, which were never meant to stand such a strain, and consequently yield to it, giving us bronchiectasis, local or general, and emphysema. If the lung trouble is of such a nature as to be associated with much scar tissue, then this may by contraction constrict air tubes, leading to dilation above the obstruction, or if it has a distant point of support its contraction may lead to a pulling open of tubes. We must not forget, too, that in elderly people, at all events, there is yet a third factor, and an important one, in the production of these degenerative or fibrotic processes; this is arterial degeneration or wearing out of arterioles, with consequent imperfect nutritional changes in fine tissues. It is a clinical fact that an enormous numerical preponderance of cases of "bronchitis and emphysema" in persons over forty, say, have atheroma of arteries well marked, and I believe the association is causative, and not an accidental accompaniment of age. We are thus led by simple deductive reasoning to appreciate the pathology and occurrence of—

The Pulmonary Complications of Chronic Bronchitis

(1) *Bronchiectasis*, of which the principal diagnostic features are: large quantities of expectoration, often brought up in mouthfuls at a time, and frequently fœtid; this fœtor is a most important diagnostic point between bronchiectasis and a phthisical cavity, for the latter never or hardly ever possesses fœtid contents; otherwise the physical signs of the two conditions are almost identical.

(2) *Emphysema*, of which hyper-resonance or too great an extension of resonance is the main sign, coupled with prolonged expiration and shortness of breath out of proportion to the cardiac condition or the signs of bronchitis.

(3) *Collapse*, arising from constriction of a tube or from plugging of it by catarrhal products. Over a collapsed area there is likely to be a little alteration in the percussion note—dulness or woodeny sound—but absence of breath sounds

will be naturally the chief feature, and, possibly, slight fine crepitus on very forced inspiration.

(4) *Hæmoptysis*, from rupture of a vessel, either owing to degeneration or to mechanical stretching, or possibly to venous distension in a violent paroxysm of coughing.

(5) *Pleurisy or pleuritic effusion*, arising from extension of inflammation or from passive disturbance of circulatory exchanges; diagnosed by a rub or dull percussion note with ægophony, etc. (*vide* below).

(6) *Bronchopneumonia and capillary bronchitis*, arising from extension of the catarrhal process to bronchioles and alveoli, either in an ordinary and direct manner, or by the insufflation of the decomposing contents of a bronchiectasis. These are perhaps the most important complications of all, for they are very frequently the direct cause of a fatal ending to the case. Moreover, tubercular bacilli may succeed, by their assistance, in implanting themselves on the weakened tissues or catarrhal contents of the tubes. The onset of capillary bronchitis (*vide* definition above) is marked either by a prominent rise (inflammatory reaction), or, more probably, by an equally prominent fall, in the temperature to subnormal (asphyxial or exhaustive decline). This alteration will be associated with a very marked exaggeration of the breathlessness and cyanosis, the pulse will speedily become extremely frequent, small, and thready, and the patient will rapidly pass into a condition of extreme danger from asphyxia; in true universal capillary bronchitis he never rallies, but cold, clammy perspiration soon appears, and death follows in less than forty-eight hours. The diagnosis of bronchopneumonia, when associated with a smart febrile attack, does not present any difficulty; but when we are attending an elderly patient, the subject of chronic bronchitis, we must be on our guard to examine the lungs pretty frequently, as this alveolar extension often creeps on in a most insidious manner (pleuritic effusion may behave similarly, *vide* below), a little extra cough and shortness of breath, a little flushing of the face, perhaps, or sense of weakness or exhaustion, being the only outward indications of the mischief, which is most serious in its result. If we do, however, discover by care such a smouldering danger, we must not forget to test the sputum for bacilli, as this is the

common history of tuberculosis supervening on chronic bronchitic trouble.

PLEURISY AND PLEURITIC EFFUSION

To discover the mere presence of these troubles is usually easy; to estimate aright their clinical significance is far more important and difficult.

Acute Pleurisy

Symptoms and Physical Signs.—Of the diagnosis of the presence of acute pleurisy I shall only say that pyrexia, associated with cough and pain in the side made worse by forced respiratory movements, is very characteristic, and if friction sounds are heard distinguishable from pericardial ones (*vide* p. 126), the diagnosis is complete. I have already (on pp. 61 *et seq.*) drawn attention to other more obscure cases of pain in the side.

Causes of Acute Pleurisy.—These may be divided into the general and the local. As regards general causation, experience shows that the following are very liable to an attack:—

(1) Those who are suffering, or are liable to suffer, from rheumatism, acute or subacute.

(2) Those who are affected with, or recently convalescent from, an attack of an acute specific fever, including influenza.

(3) The victims of chronic nephritis of any form, with its associated imperfect elimination of waste products, and its anæmia.

(4) It is undeniable that a fourth group must be inserted of individuals who are apparently in perfect health, but get an attack of pleurisy as the result of what is ordinarily termed a chill. Recent observations on the subsequent history of such individuals tend to reduce the number of genuine idiopathic cases very materially, as a large proportion of them have proved to be concealed tuberculosis.

The *local causes* are:—

(1) Traumatism, blows, fractured ribs, crushes, etc.

(2) Extension of inflammatory processes—

(*a*) From the lung, tubercle, pneumonia, bronchopneumonia, infarction, abscess, gangrene, etc.

(*b*) From the pericardium (rarely).

(*c*) From abdominal viscera, liver, spleen, etc., through the diaphragm.

(3) Rupture of morbid collections into the pleura, independently of the inflammatory process reaching it—phthisical cavities, abscesses, hydatids, etc.

Pleuritic Effusion

Symptoms.—Inasmuch as effusion may be, and frequently is, an associate or sequel of the acute affection, however arising, the symptoms of acute pleurisy will suggest an examination for fluid; but, on the other hand, effusion frequently takes place very insidiously, without obvious precedent disease or symptoms of sufficient severity to induce the subject to take medical advice, and only a troublesome shortness of breath or vague chest discomfort leads him to submit to examination.

Physical Signs.—On the affected side we find:—

On inspection.—Some loss of mobility compared with the sound side, possibly a visible displaced heart's apex beat.

On palpation.—Tactile vocal fremitus, absent or much diminished, possibly heart's apex beat felt in wrong place.

On percussion.—Absolute dulness with increased resistance to the pleximeter finger.

On auscultation.—Weak or absent breath sounds, probably of a distant tubular character owing to compression of alveoli and finer tubes; a peculiar nasal twang is given to the voice sounds, known as ægophony. Heart sounds heard most clearly in a displaced position.

Over the still uncompressed lung the sounds, *qua* effusion, will be healthy, but the breath sounds will be exaggerated from overwork.

Causes of Pleuritic Effusion and Hydro-Thorax:—

(1) All the causes of acute pleurisy already enumerated, in any of which effusion may be a predominant feature, or may remain after the acute symptoms have subsided.

(2) Heart disease, with its obstructed circulation.

(3) Malignant disease and other growths (*e.g.* hydatid) within the thorax.

(4) Tuberculosis of lung or pleura, either evident or concealed (*vide* Causes of Acute Pleurisy).

A general review of the above paragraphs leads to an inevitable conclusion that pleurisy and pleuritic effusion must in almost all cases be reduced to the rank of a symptom, possibly it may sometimes rise to the dignity of an independent disease, and as a symptom it may occasionally possess the importance of causing a fatal event; but, on the whole, its features and pathology must be analysed from the point of view of what lies behind, and not as the "be all and the end all" of a diagnosis.

The Nature of the Fluid.—Physical signs alone enable us to say that fluid is present, but they give no direct information as to its nature—unless, indeed, we have a pointing empyema, which is, or should be, very rarely seen nowadays. Indirectly, by their persistence, they tell us that the fluid is not being absorbed, but this may be, and generally is, due to circumstances other than its nature. Should there be no indications of these circumstances, such, I mean, as obvious morbus cordis or pneumonia, etc., we are then justified in saying that this non-disappearance is probably due to the fluid not being simple serum, or to there being some hidden and persistent cause for it in the thorax, *e.g.* tubercle or carcinoma; cases of simple idiopathic pleuritic effusion resisting absorption for any length of time are rare. The symptoms, again, strictly speaking, are not of much use: excessive sweating, anæmia, weakness, and hectic temperature are certainly suggestive of pus, but they do not exclude tubercle with simple serous effusion, nor, indeed, carcinoma, which I have found on the post-mortem table after such precedent history. There is but one method of ascertaining with certainty the nature of the fluid, and that is to withdraw a little by a syringe and examine it with the naked eye and (or) microscopically.

The following considerations will show that even now our diagnosis is far from complete: the fluid may be (1) clear watery, (2) bloody or blood-stained, (3) milky or purulent, (4) stinking.

(*a*) If it be clear, it is either hydatid-cyst fluid—which will not coagulate on boiling and will show hooklets—or it is serum; in this latter case no strong deduction can be made from the information, for every single cause may have at times clear effusion, except ruptured abscesses and gangrene, both of which have probably given other and clearer proofs of their presence. It is in clear effusions that inoculation experiments on animals are so useful in clearing up a doubtful tubercular diagnosis.

(*b*) If it be turbid or definitely purulent, the suggestion is that the attack is strong or the defence weak, which is not very

conclusive information; empyema is common in children and in old people without further obvious cause, and also in renal cases and drunkards, but I have met with it as the product of carcinoma.

(*c*) Blood or blood-stained serum is naturally to be expected in severe traumatism or ruptured aneurysm, in purpuric or scorbutic patients, but when occurring without such immediately obvious antecedent, it certainly leads us strongly in the direction of tubercle or carcinoma, for it is very rare in simple or rheumatic cases.

(*d*) If it be stinking, we have conclusive proof that the microbes of decomposition have gained access to the fluid, but how, or why, they got there, we must learn from other evidence than their presence. Some cases of stinking empyemata unquestionably do perfectly well after simple drainage, proving that the access of the microbes was not gained by gross lesions.

It will thus be seen that the nature of the fluid *per se* gives but little definite or decisive indication[1] as to the real meaning of pleuritic trouble; serum clear or blood-stained, pus, or stinking fluid may each apparently occur or succeed one another in the same case; the point is of great importance for prognosis and treatment, but we must look elsewhere for clearer indications of causation. The presence of acute rheumatism, or the history of its previous occurrence, an account of severe exposure to weather, a present or recent attack of an acute specific fever, of pneumonia, of bronchitis or bronchopneumonia, advanced tubercular changes in a lung, are all fairly obvious on slight inquiry or observation. So, too, heart disease or chronic nephritis will yield unmistakable evidence of their presence, if we only bear them in mind as possible causes and look for them. Any of these would be admitted as a satisfactory and final explanation of the origin of the trouble, beyond the natural history and clinical significance of which we need not go either for diagnosis or prognosis; but if the case offers no such leading indications, we have now to decide between (1) an unusual case of apparently idiopathic pleural effusion; (2) malignant growth; (3) tubercle of lung or pleura concealed by the fluid. If the fluid has persisted, without signs of absorption, for longer than, say, two or three weeks without a causative diagnosis becoming reasonably certain, we should, I think, empty the chest as completely as possible by mechanical means, even in the absence of urgent symptoms or other reasons, solely for the purpose of establishing, as far as

[1] Microscopic or bacteriological examination may possibly in some cases give absolutely conclusive proof of tubercular or malignant disease.

possible, a fundamental diagnosis from the nature of the fluid, and from the changes in the chest produced by its withdrawal. The nature of the fluid we have already seen to be very dubious in its indications, though *something* may be guessed from it. The changes in the physical signs may be thus analysed:—In simple idiopathic pleurisy aspiration will certainly cause a restoration of some degree of resonance to the previously dull area, and the lung will soon expand with a return of distinct breath sounds, probably intermingled with fine and coarse crepitations, owing to the renewal of the circulation through previously compressed lung tissue. If the case is a tubercular one, it is again probable that there will be some restoration of more natural physical signs on percussion and auscultation, but it will not be so complete as in the former case, and there is likely to be some evidence of patchy consolidation, and quite possibly friction sounds will be heard. It is, too, more probable that the fluid will return, though in either case this may happen. In growth of more gross character it is unlikely that the removal of the fluid will alter to any marked degree (*vide* below) the physical signs found previously; there is no re-expansion of lung with renewed air entry, rapid return of the fluid is practically certain to take place, and suspicion becomes greater the more rapidly this return appears without pyrexial phenomena to account for it.

If the fluid withdrawn be turbid or purulent, this is in itself strong, but not absolute proof against malignant growth. The most important point to bear in mind with pus is that the fibrinous coagula, etc., are apt to prevent expansion of the lung, so that greater care must be exercised in pronouncing judgment from the absence of this expansion. It is also very rare—I should say unknown—that tubercle, *otherwise indetectable*, should cause a purulent effusion as the first sign of its presence.

INTRATHORACIC TUMOURS

Those which I propose to discuss here are: (1) malignant growths; (2) aneurysms. Both have their chief features in common, viz. irritation and pressure, and to discuss in detail every variety of each, and its separate diagnosis, would require a monograph to itself. I only propose to give the principles on which such differentiation must proceed; this necessitates as a foundation a brief sketch of the nature and anatomical relationships of the two affections.

Malignant Growths

Are in nature—

(1) Primary.
- (*a*) Carcinoma.
- (*b*) A group to which we may loosely apply the term "lymphosarcoma." [1]

(2) Secondary. Every growth or form of it which can cause metastatic deposits, or invade the thorax by simple local extension.

Their anatomical relationships may be thus tabulated:—

	Starting Point.	Progress.
Primary.	Almost invariably at the root of the lung, either in the root itself or glands of posterior or upper mediastinum.	1. Into the mediastina, surrounding and compressing the structures there, and forming a large solid mass.
Secondary.	(*a*) Anywhere in the lung tissue. (*b*) Creeping into the mediastina.	2. Into the substance of the lung, solidifying it. 3. Into and over the surface of the pleura pericardium, etc.

I have grouped these together, allowing a growth to proceed in any direction, a course which is theoretically true, and may now and again be taken by any of them, but clinical experience in the post-mortem room shows that:—

1. Primary carcinomata almost always spread either into the lung tissue or on to the pleura, or both; they practically never invade mediastinal tissues alone.

2. Primary lymphosarcomata practically never *invade* either lung or pleura, but grow steadily in the mediastina, *compressing* the lung; the pericardium is surrounded, and may be actually invaded on its inner surface, causing effusion and cardiac difficulties.

3. Secondary metastatic deposits from a distance practically always form nodules in the lung substance, *solidifying and invading it without compression.*

4. Secondary extensions from mamma or œsophagus, *e.g.*, are the especial tumours which may spread in any direction.

[1] This term must include true sarcomata, and also the ill-understood enlargements of lymphatic glands, lymphadenoma, Hodgkin's disease, etc.

Aneurysms

May be classified according to the part of the arch from which they spring, but I think more usefully according to the clinical indications, thus :—

Anatomically.	Clinically.
Of the ascending arch. Of the descending arch. Of the descending thoracic aorta.	= The aneurysms of physical signs especially : (*a*) Externally bulging tumour with expansile pulsation. (*b*) Dulness on percussion. (*c*) Audible bruits.
Of the transverse arch.	The aneurysms of symptoms, principally or entirely pressure effects on neighbouring soft tissues (*vide* p. 108).

The progress of any aneurysm—upwards, downwards, forwards, or backwards—is governed by the precise point in the circumference of the vessel at which it first starts, spreading from that point in all directions except towards the parent vessel.

This sketch shows that the two conditions cannot be separated by anatomical considerations alone. We will now discuss in parallel columns the more likely explanation of certain conditions.

	Aneurysm.	Growth.
Sex.	Many times commoner in males.	No predilection for either sex.
Occupation.	The more laborious occupation probably explains the greater frequency in men.	Would seem to have no influence in inducing a growth primarily.
Age.	Usually thirty-five to fifty.	Primary commoner under thirty-five, but secondary at any age corresponding to the primary growth.
Alcohol.	Excess undoubtedly a predisposing factor.	Without influence.
Syphilis.	Is undoubtedly also a predisposing factor.	Has no influence on ordinary growths ; and gummata, large enough to cause doubt, are excessively rare.

	Aneurysm.	Growth.
Complaint of pain.	Acute pain is certainly suggestive in cases of doubt, but no conclusive evidence from this point.	If present it is more commonly a dull oppressive pain, but neuralgia may occur.
Inspection of patient.	Bulging expansile tumour pathognomonic; but, of course, usually no evidence from inspection in cases of doubt. Œdema of an arm occasionally seen, but of one side of chest *alone* very rarely if ever.	A mass of enlarged glands or growth above clavicle also pathognomonic; a malignant tumour elsewhere is very suggestive in doubtful cases. Œdema of *one* side the chest wall very suspicious, and if *without* œdema of arm almost conclusive.
Auscultation.	A systolic or double bruit, if not traceable to valvular disease, is very suggestive; even if valvular it only loses significance if patient be young and has suffered from a recognised cause of endocarditis. The degenerative processes of age tending as much to aneurysm as to valve destruction.	Bruits have no connection with tumour as such; may occasionally occur if heart is displaced, or aorta or pulmonary artery distorted, but this is very rare.
	Pressure and Invasion Effects	
On lungs.	Usually causes compression only, with irritating cough, but may start an acute inflammatory attack with free hæmoptysis.	May cause compression only by growing on the pleura (*q.v.*), but more frequently assumes the characters of an insidious consolidation — often multiple in secondary infection — and only at the last when breaking down causes aspiration-bronchopneumonia.

	Aneurysm.	Growth.
On trachea and bronchi.	Tracheal tugging[1] very characteristic if present; may cause a bronchitis, and possibly dyspnœa, but *vide* Nerves.	Probably causes only a slowly progressive shortness of breath from compression, which also leads to collapse of corresponding area of lung.
On pleura.	Acute pleurisy a much commoner result than quiet effusion; this is because an aneurysm is much more acutely irritating than a solid neoplasm.	A clear or blood-stained effusion, very common when the growth reaches to and spreads over the pleura; it is probably more due to venous blockage than a real inflammatory reaction.
On veins.	Aneurysm, until too large to be mistaken, rarely presses on any vein except the left innominate, and hence the œdema mentioned under Inspection not usually available in doubtful cases.	Tumour of size or extent to be still doubtful frequently catches the intercostal veins, causing œdema of chest wall; also the vena azygos, causing enlargement of abdomino-thoracic veins; the left innominate, too, or even a vena cava, may be caught early.
Thoracic duct.	Practically never interfered with by a *doubtful* aneurysm.	Is now and again blocked by a growth, and occasionally chylous ascites has appeared from this cause.
On œsophagus.	Aneurysms cause dysphagia, but when it arises from this cause it is likely to vary in degree on occasions.	Tumours of thorax proper more rarely than aneurysm cause dysphagia; should they do so its amount is likely to be unvarying. Carcinoma of the œsophagus itself is the only likely one, and then the gradual but persistent history of

[1] A peculiar shock synchronous with the pulsation of the aneurysm, felt by two fingers laid on the trachea when extended to its utmost by throwing back the head as far as possible.

	Aneurysm.	Growth.
		dysphagia as the *only* symptom is the strongest circumstantial evidence.
On nerves.	The intermittent beating of an aneurysm appears to be more irritating than a growth; the recurrent laryngeal is the nerve *par excellence* of aneurysmal trouble, being frequently paralysed by small aneurysms of the arch.	A growth appears to cause but little active irritation to nerves, but no general rule can be laid down. Pain is certainly less common in growth than in aneurysm.
On bones.	Destruction of bone by aneurysm is very painful.	Intrathoracic growths rarely reach and erode bones, but one growing from the bones of the thorax would cause pain not so acute as that of aneurysm.
Effect of rest or treatment.	The symptoms of aneurysm show spontaneous variations in intensity which it is difficult to account for; they are almost invariably improved, for a little while at all events, by rest in bed and other treatment.	The symptoms of growth rarely show even the faintest trace of improvement under rest and treatment, while judgment is suspended.
Duration of symptoms.	From clinical histories it would appear certain that aneurysms may last for a long time before causing a fatal event, hence a history of symptoms for many months or a year or two is very suggestive of aneurysm as against growth, though perhaps more against either of them.	All cases of definite tumour end fatally within a year, hence if condition has early and rapidly become severe, it is so far in favour of growth *v.* aneurysm.

These points, I think, include the bulk of the evidence we shall have to weigh in a given case. Few only of them are decisive or pathognomonic, but if all are carefully considered I think we shall usually arrive at a correct conclusion.

CHAPTER IV—*Continued*

DISEASES OF THORACIC ORGANS

SECTION II.—DISEASES OF THE HEART AND PERICARDIUM

ERRORS of judgment—if not of actual diagnosis—occur more frequently in connection with heart disease than with the troubles of any other organ. The reason is somewhat as follows:—Before the days of the invention and perfection of the stethoscope heart disease was either not diagnosed at all, or it was guessed at by pain and morbid sensations in the thoracic cardiac area, or it was recognised by evidences of circulatory disturbance and the character of the pulse. These indications, without the assistance afforded by the stethoscope, were apt to be misleading. Now the pendulum has swung too far the other way, and, armed with an instrument of precision, we are all too apt to think we can immediately diagnose heart disease by bruits thus brought within the sphere of our objective consciousness. This is just as great a mistake as the former condition of uncertainty. That we can thus detect a leaking valve or a stenosed orifice must in general be admitted—though even here great caution is necessary in interpreting our aural perceptions—but this is often enough a very different matter from morbus cordis. Sir Andrew Clark was, I believe, the first to draw public attention to this. He compiled a very long list of patients in whom he had by systematic examination detected a cardiac murmur, but whose symptoms and complaints, as he showed, had no derivation whatever from a diseased heart. His experience we all of us can now confirm from out-patient practice, but it seems somehow to have escaped the recognition it deserves at the hands of students and practitioners.

It may then well be asked what are the features (symptoms

and physical signs in combination) by which we are to recognise genuine heart disease. The conditions leading to a suspicion of heart disease are divisible into two main groups: (1) local in the thorax or cardiac area; (2) general, expressed in or by distant organs or tissues; and each may again be divided somewhat indefinitely into symptoms and physical signs (p. 21). We will proceed to comment briefly on each division.

Local Thoracic Symptoms which may Indicate the Presence of Morbus Cordis

1. *Pain.*—Pain is a very variable feature indeed. It may be slight, or it may be extraordinarily severe (typical angina), and almost any degree of it may be associated with a cardiac condition of absolutely no moment at all, or of one of the very greatest danger to life. The only general statement that can be made about it is, that when due to the more serious troubles, it is very liable to spread from its original position and extend to the right, or to the upper chest and down the left arm. On the other hand, when owning a gastric (its most common source) or other than cardiac origin, it more commonly remains fixed to the cardiac area. It must not be forgotten that the pain of aneurysm, of pericarditis, and of left pleurisy, all instances of serious organic disease, more commonly also remain as a stationary pain. In any and all cases we must, however, make a careful physical examination, for by the results of this our judgment of the pain is, after all, mainly influenced. Pain alone must not be accepted as evidence of morbus cordis, though, when we are satisfied that the pain is genuinely severe, we must be doubly cautious in exonerating the heart, even if at the time of examination there remains very little or no evidence of abnormality.

2. *Shortness of Breath* may be very briefly dismissed, inasmuch as it may be due either to pulmonary or cardiac conditions, and it is only a physical examination of the patient that can determine which. From either source it is probably associated with cough, and made worse by physical exertion. That produced by cardiac disease is perhaps more definitely relieved by raising the patient to a sitting posture.

3. *Cough.*—Cough perhaps more properly belongs to the general group of back pressure symptoms, but as its alternative causation is local, it may be mentioned here. If due to cardiac disease, it will be either dry, ineffectual, and useless cough, and is then

suggestive of nervous irritation, perhaps aneurysm, or it will be accompanied by some expectoration, but this will be simple frothy mucus, unless, indeed, there be hæmorrhage, when the mucus will be blood-stained or replaced by pure blood (*vide* Hæmoptysis, p. 54). If pus or other organic elements be present, it is a certain demonstration that the cough is not *purely* cardiac in origin.

4. *Palpitation.*—This simply means that the beat of the heart has become perceptible to the owner of the organ. It is extremely common in all sorts of diseases and conditions of ill-health, and especially as the result of mere introspection and morbid self-consciousness. Accordingly, when standing alone, without objective signs of morbus cordis, it is almost an indication against that condition as its real source; but when associated with definite abnormalities of mechanism or rhythm, and especially if then constant, it acquires considerable significance in the case.

We thus see that the local symptoms, without exception, require a careful physical examination to determine their value and significance.

Local Thoracic Physical Signs which may indicate the Presence of Morbus Cordis

These include everything which can be seen on inspection, felt by palpation, appreciated by percussion, or heard on auscultation. I do not propose to discuss these in any detail, but shall merely indicate *seriatim* what aids they give us in diagnosis.

Inspection is very frequently quite negative in its results. It may show a heaving impulse of great hypertrophy, or the pulsating tumour of an aneurysm; the apex beat, too, may be visible to the eye, and its position is of great importance (*vide* p. 102 for its use in pulmonary diseases). A rippling wave of apex beat is suggestive of irregular action, or possibly of pericardial effusion, and recession near it in systole might possibly indicate adherent pericardium.

Palpation will further determine the apex beat and be convincing of a strong, heaving beat. It may rouse suspicion of irregular heart action. Stress is laid by some on a thrill which may possibly thus be felt. It certainly is corroboration of probable mitral stenosis when a doubtful presystolic bruit is heard, but is frequently felt with failing heart from any lesion, and also may be appreciated often in thin subjects whose hearts are above suspicion, so that I hold it very cheaply unless other and more certain indications of trouble are present.

Percussion gives, with very variable results, the area of uncovered heart, and possibly some idea of the gross size of the organ. But it is so subject to personal variations in the observer, so liable to vary in health in different individuals, so subject to fallacies from the position of the lungs in health and disease, that it is wise to reduce it to a very low level of diagnostic utility. The preposterous claims made on its behalf by the recent upholders of the Schott (and other) systems of treatment have reduced its value still lower, and left it as an object of ridicule to thoughtful men, at any rate as indicating the size of the heart. It is, however, of some little use in helping us to differentiate between a pericardial effusion and a hypertrophied heart.

Auscultation.—By auscultation we become aware of several factors in the heart beat which are of very unequal significance individually, but collectively constitute our most important knowledge of the condition of the heart. They may be enumerated as (1) the ordinary sounds of the heart; (2) the frequency of the beat; (3) the regularity of cardiac action and its rhythm; (4) abnormal sounds or bruits added to or replacing the natural sounds.

(1 and 2) *The Ordinary Sounds and Frequency.*—In health it is well known that the first sound should be materially longer than the second, the now classical lubb-dup well representing the difference. As the frequency of the rate increases this difference must, of course, *absolutely* diminish, and in rates of over (say) 150 is practically inappreciable; but when moderate or low frequency is present, even in the absence of other abnormality, a comparative shortening of the first sound is a serious indication of some change in the action. It may indicate mere nervous hypersensitiveness, and then the rate is likely to be high, and other evidence of nervous instability may be present in the patient. More usually, and especially with moderate frequency of beat, it is suggestive of muscular debility or hurry, probably the result of degeneration, or, at least, of imperfect nutrition, and makes us anxious about the supervention, already present or to come, of a pathological yielding of the muscle and incapacity to continue to stand the strain of present pressure.

(3) *Regularity.*—So long as the action of the heart is regular, and its rhythm even, so long may we make a very substantial deduction from the prognostic gravity of almost any evidence of defective mechanism or circulatory disturbance. If irregularity be detected especial care must be taken to ascertain if it be a regular periodic irregularity, *i.e.* an occasional dropped beat say every four,

five, or six beats, or if it be a totally irregular irregularity, defying exact description, and rhythm totally absent. The former is often a harmless condition, and may even be beneficial to a heart working under some unusual strain, so that in the absence of other definite indications of cardiac failure it requires only to be noted for future reference, but is not to be treated by direct efforts at cardiac medication. The latter, a tumultuous irregularity with many beats of very variable strength, is a very grave sign, and indicates the urgent need of prompt interference on behalf of an overburdened heart.

(4) *Abnormal Sounds.*—The presence of an endocardial bruit (*vide* p. 127, to differentiate endo- from exocardial bruits) heard in the situation and conducted in the direction usually laid down in text-books, is strong evidence of leakage through, or narrowing of, a valvular orifice, but the deductions as to cardiac disease to be made from this evidence are of very unequal importance in different cases. In rheumatic fever or other disease, such as the specific fevers, known to be a possible cause of endocarditis, *the first occurrence* of a bruit is of very great importance, for we then know that a valve is inflamed and softened, and we get a strong indication for giving such valve as much rest as possible. Again, in a case of smouldering but persistent pyrexia, the source of which is not obvious, if we hear a cardiac bruit which was not present before, or even if we were previously aware of its presence, but it seems to vary in character at times, it will throw strong suspicion on that valve as the seat of a septic or simple inflammation, keeping up the temperature and threatening very serious danger. Thirdly, when some of the features of circulatory disturbance through an organ or district are present, a bruit is certainly suggestive of central cardiac failure as a probable contributory factor, though regularity and evenness of the cardiac beat are much more important features than the bruit itself. In the more chronic cases of valvular trouble, where a bruit or bruits have been known to exist for some time, neither the bruit nor the precise form of trouble indicated by it is of great importance, for we can neither treat the one nor place any great reliance on the accuracy of the other. Speaking then in general terms, bruits are only of value to remind us (1) when they first appear, they are evidence of a softened valve which may yield on pressure; (2) when old, that the heart is working with imperfect machinery, which has not the same elasticity for emergencies that the original sound mechanism possessed.

General Symptoms in other Organs which may indicate Morbus Cordis

There are at least two factors in the production of those symptoms which are exhibited by other organs or areas, but which are usually accepted clinically as indicative of cardiac disease. They are: (1) the work of the heart and aorta; (2) the work of the smaller arteries, arterioles, and capillaries interacting with the lymphatic circulation through the part in question.

The heart is a driving and suction pump, introduced into the closed system of tubes, by means of which the blood is kept circulating, and as such its nervous, valvular, and muscular mechanisms are perfectly adapted for fulfilling its part in the total result, viz. that of keeping up a sufficient head of pressure in the aorta and pulmonary artery, and relieving by suction the largest venous trunks of their low pressure contents. The aorta in turn, distended and stretched by this charge of blood at high pressure, should, by its elastic recoil, be able to transmit the charge and pressure in gradually diminishing degrees to the arteries smaller than itself. The greater or less frequency of the heart beat, and its greater or less force, are regulated by the central nervous system only according to the very general needs and conditions of the body at large as a whole. It is to the second of the above factors, viz. arteriolar, capillary, and lymphatic action that we have to mainly look for the regulation of the blood supply for the *local* needs of individual organs for particular purposes, and also for an explanation of the finer problems of local nutrition and function. That this is in many cases the most important factor is strongly suggested, if not proved, by cases where death has taken place with the patient in a water-logged condition, and yet post-mortem examination has shown the heart itself healthy enough, in both muscle and valve, to keep up its part of the circulation for a long time if local conditions had allowed or assisted their share of work to be done. It must be admitted that improvement in the heart's action is frequently followed by the happiest possible results in distant parts, so that it is impossible in clinical medicine to separate and identify the exact shares of each factor; none the less the effect of digitalis or suprarenal extract on the one hand, and of iodide of potassium, nitroglycerine, or erythrol tetranitrate on the other, compel us to bear in mind the peripheral as well as the central circulatory factors. Whichever of the two is at fault, or if both be so, the ultimate local

result from a clinical point of view is much the same. It is to be remembered, too, that the precise nature of any valvular defect influences only *the time* when circulatory disturbances appear, and *then the order or sequence in which* they occur; it has no *general* influence on the result, provided that cardiac muscular failure actually supervenes. The result itself is, in the first place, a diminished arterial supply of fresh blood, together with an imperfect or inadequate removal of the impure venous blood, and renewal of nutrient lymphatic plasma. This circulatory disturbance leads in its turn to interference with the work of organs with an active function, and to œdema or effusion in areas of less functional activity. In the second place, this sluggish capillary venous and lymphatic circulation, with excessive pressure on the venous side, leads to an alteration in the quantity and quality of the natural exchanges between blood capillaries and lymphatic channels, and, in fact, alters *in toto* the vital conditions existing in the part in question. Thus we find not unfrequently that a venule actually ruptures with gross effusion of blood, or individual red cells escape and die in an extra vascular situation, or finally the blood may coagulate in the venules from unnatural composition, leading to a condition known as infarction. Ultimately, but gradually, fibrin coagulates from the altered lymph, and causes a thickening of the capillaries and fibrosis of the tissues, and the pigment from the dead red corpuscles stains the tissues. Again, we have embolism occurring, not as the result of general circulatory conditions, but owning a particular cause in either loosened vegetations from endocarditis, or clotting of blood in the irregularities of a cardiac chamber, with subsequent loosening of the clot into the circulation. The symptoms occurring in these various ways throughout the body may be tabulated briefly as follows:—

Organs.	Symptoms of back pressure.	Post-mortem appearances known as "heart" organs.
Lungs.	Shortness of breath, cough dry or with simple frothy mucous expectoration, moist sounds at one or both bases. Infarction or embolus leads to bloody sputum, and prob-	Drier than usual and tougher, more resistant to attempted laceration by finger; probably discoloured from old blood pigment (brown induration); round or wedge-shaped area

Organs.	Symptoms of back pressure.	Post-mortem appearances known as "heart" organs.
	ably pleuritic pain and rub with physical signs of consolidation: this used to be termed pulmonary apoplexy.	of consolidation = infarction or embolus.
Pleura.	Pleural effusion common, with appropriate physical signs.	Excess of fluid in the cavity.
Liver.	Tender or even painful, and somewhat enlarged to the examining hand.	Congestion of intralobular veins, well-known nutmeg liver.
Spleen.	Possibly enlarged, may be vague pain in region.	Very firm, may be small or large, and possibly wedge-shaped areas of embolism.
Stomach and intestines.	Dyspepsia, flatulence, and constipation frequent; severe vomiting often seen.	Veins very prominent, and mucous membrane dark and congested; possibly slight ulceration.
Kidneys.	Diminution in secretion even to almost complete suppression; slight albuminuria very common.	Kidneys very firm on section; cortex equally congested with the medulla.
Brain.	Headaches, confusion of mind, sleeplessness, noises in head, flashes of light, etc.; apoplexy of some form common enough.	(?) serous apoplexy; brain often *seems* more juicy than normal. Embolus a well-known cause of softening.
Limbs.	Pitting œdema, especially in the legs. Emboli of arteries.	

It is particularly important to remember that any organ whose vitality and circulation are thus interfered with is in a condition in which very slight irritation will cause an acute outburst of inflam-

mation. Bronchitis and acute nephritis are thus two very common complications, and it is often a matter of some importance, but great difficulty, to determine whether the bronchi and kidneys are simply venously congested or actively inflamed. I am accustomed to rely upon a muco-purulent *v.* mucus sputum, and the presence or absence of dry musical rhonchi in the one case, of epithelial casts with facial œdema in the other case, as the most reliable points of distinction.

Such, then, are the means we have for determining the presence of real heart disease, and, like similar evidence on any other point, each factor has to be weighed first *individually*, and then in addition to, or subtraction from, all other factors, and a balance struck with every possible care.

The matter will be referred to again in dealing with nervous *v.* valvular and muscular heart troubles (*vide* pp. 137 *et seq.*).

We may now consider the pathology of, and the meaning of, the terms—

HYPERTROPHY AND DILATATION, COMPENSATION AND FAILURE

Hypertrophy

It is a provision of nature, distinctly tending to the preservation of individuals, that healthy muscle should increase in volume and strength (growth or hypertrophy) in proportion to the work it is called upon to do, *provided that this at first lies within the original capabilities of the muscle.* The heart is no exception to this rule, and when its circulatory work from any cause (nervous, valvular, or connected with increase in general blood pressure from kidney disease) is *persistently* maintained above its original average, the cardiac muscle hypertrophies or grows to meet the demand, provided that the nutritional powers in general of the individual are fairly good. Temporary increase in work is inherently provided for by a reserve excess of potential energy over that habitually put forth.

It is easy to understand in general terms that if the mechanism of the heart was originally adapted to work with the greatest possible efficiency (and we have every reason for such belief), any alteration in that mechanism must involve increased expenditure of energy to produce the same result, *i.e.* increased work. We have just seen in the previous paragraphs that the symptoms of disease of the heart as expressed by organs other than itself are largely (the

vasomotor factor must not be forgotten) indicative of failure of the heart to do this increased work. All the means which unaided nature can take to bring about a fresh balance of work and power are collectively spoken of as COMPENSATION, and hence compensation clinically means regained (if lost) or maintained capability of the heart to do its work under unusual or altered conditions. For a *permanency* this is, to all intents and purposes, hypertrophy. *Temporary* natural compensation was mentioned above as a mere use of potential inherent energy, and artificially we can produce temporary compensation by diminishing the work, as by rest in bed or vasodilator drugs.

Following this argument, it is obvious that hypertrophy has only one cause—increased work. It is actually the result of, and can only be equal to, this increase, and then it must logically be deduced that the symptoms of hypertrophy are—the absence of symptoms of heart disease. Disappearance of these symptoms when present means the development of compensation, and *if permanent* this means hypertrophy.

It must be admitted that there are circumstances under which hypertrophy may be indirectly the possible cause of some trouble. If, for instance (as in athletes, soldiers, etc.), great physical exertion has called forth considerable hypertrophy, then, when the work is permanently given up, the heart probably will not diminish in bulk in proportion to its diminished duties, and its beat may become inconveniently apparent to its owner. It is possible that its powerful action may rupture an artery that is healthy. Certainly it is a source of danger to vessels that are not healthy, and arterial disease and degeneration are unfortunately only too frequent results of the same conditions as those which require hypertrophy of the heart. In this sense hypertrophy of heart may be described as a predisposing factor in cerebral hæmorrhage, but in all other senses hypertrophy is a purely defensive expression of nature—a physiological and not a pathological phenomenon.

DILATATION

We may say at once that this term is used in two distinct, and in some respects almost opposed, senses. The first is a mere mechanical increase in the capacity of a chamber. The second sense takes note of only one of the possible consequences of this increased capacity, and is the equivalent of cardiac failure.

As regards the first meaning, theoretical considerations would

show that it is an inevitable and universal result of valvular incapacity, though I am strongly inclined to doubt if it is possible to *prove* the position by post-mortem evidence (the conditions of rigor mortis, and the effects of pressure upon dead muscle, do not sufficiently closely correspond to the reaction of living muscle to its environment); anyhow, the occurrence of dilatation in this sense has no possible importance from a clinical point of view, *provided that the energy of contraction and the strength of the chamber in question are increased in proportion*,[1] the object of the muscular walls being merely to empty the cavity with sufficient force.

The second sense in which dilatation is used is exactly represented by the absence of this requisite energy of contraction and absolute strength, and it is precisely this that is the cause of the symptoms of morbus cordis. This is heart failure, or absence of compensation. It is under these circumstances that a previously hypertrophied heart becomes a nuisance to its possessor by its tumultuous efforts to cope with the work to which it is no longer equal. It is when this failure or stretching becomes excessive that relative incompetency of healthy valves supervenes, for they are no longer capable of closing an orifice which has become too large.

DIAGNOSIS OF HYPERTROPHY AND DILATATION

As the presence of one or other or both of these conditions is, one might say, the very essence of 99 per cent of cardiac pathology, the recognition of them becomes at once the cardinal point in diagnosis.

Of dilatation in its first or simple sense there is no positive objective sign at all, and its presence is a matter of no moment if the requisite muscle strength is present to compensate it. Its presence is assumed in all cases in which a large heart is due to valvular alterations; in other cases (*e.g.* that due to renal disease—so-called concentric hypertrophy) it is probably absent.

In all cases, and in any sense of the word, the condition of dilatation, as well as that of hypertrophy, demands for its presence a heart larger than usual (*i.e.* for an average environment of age, work, etc.), and therefore we require as the first element in diagnosis the signs by which we may recognise an enlargement of the heart.

[1] It must be remembered that this proportion is a large one, viz. the cube of the figure representing increase in diameter.

The ordinary physical signs by which this is done, and the principal fallacies, are the following:—

	Fallacies.
Inspection.—The apex beat may be seen displaced outwards, or downwards and outwards. A lifting of the whole cardiac area of the chest may be seen at each beat, or possibly a persistent bulging.	These physical signs found by inspection, palpation, and percussion may be:—
Palpation.—The displacement of the apex beat may be felt, as well as the heaving. If a thrill is felt it is also a suspicious sign, and must be noted.	1. Obscured—as by abundant fat or muscle over the chest, by emphysematous lung, by pericardial effusion.
Percussion.—There will be increased area of cardiac dulness, both absolute and relative.	2. Rendered unduly prominent by wasting of fat or muscle, by retraction of lung, by deformity (pigeon breast, Pott's curvature, etc.) of the chest.
	3. Their interpretation may be a mistaken one, in that tumours or effusions, etc., may displace the heart, its apex beat, and cause transmitted pulsation and bulging.
Auscultation will confirm other indications, or decide by itself the position of the apex beat. If bruits of organic origin (*vide* p. 129) are present, and known to have been so for some time, the presence of a heart larger than usual is at once certain. If no bruits are present the character of the two sounds must be noted. They are chiefly of use to separate pure hypertrophy from dilatation or failure.	Auscultation is interfered with by much the same circumstances as the other methods. But, in addition, we have to differentiate heart sounds from those of pericardial, pulmonary, and pleural origin (*vide* p. 127).

If by these means we have ascertained that a large heart is present, the only other problem requiring solution is this: Is it capable of continuing to do its work or not?

In by far the greater number of cases the answer is obvious at a glance. The symptoms of failure are too obtrusive, or, on the other hand, the absence of such symptoms is quite complete; but, excluding the extremes, we meet with many cases in which the most careful attention to every feature *may* still leave us in doubt.

Such, for example, as a young woman, possibly pregnant too, complaining of some puffiness of the ankles at night, and we find her anæmic and the possessor of a mitral bruit (the relative shares of anæmia and heart and pregnancy require much consideration); or, again, an elderly patient with renal trouble complaining of shortness of breath—Is it cardiac or renal, is the heart *beginning* to fail?

By attention to the following considerations we shall not complete our diagnosis, but we shall at least get a strong body of evidence condemning or acquitting the heart of being *particeps criminis*:—

Inspection, *Palpation*, *Percussion*: Will none of them help us much. They will reveal frequency and irregularity, but these are much better determined by auscultation.

Auscultation will give us accurate information on—

1. *Frequency of Pulse.*—It is not the absolute frequency that is of so much importance. A weak and failing heart may be either slow or frequent, fatty hearts are often slow, and sound hearts often beat over 90 per minute. What is of importance is the rapidity with which the heart settles down after a temporary increased frequency. The patient should be made to exert himself smartly for a few moments, and then the number of beats in the next four or five quarters of a minute should be registered and compared. If the muscle is good the numbers will rapidly diminish with each succeeding fifteen seconds, till the rate is regained which was previously present. If the increased frequency continues it is a suspicious circumstance against the muscle.

2. *Irregularity of Beat* in a large heart is very suspicious, and if due to muscle failure will, I believe, be invariably associated with grave features which cannot be misunderstood. It is only in hearts of average size that its indication is doubtful, and then, I think, it makes one suspect the nerves rather than the muscle in any case where there is room for genuine doubt as to the presence of heart failure (*vide* pp. 117 *et seq.*).

3. *The Character of the Sounds.*—If the natural heart sounds be replaced entirely by bruits we lose the assistance they usually afford us, and we must rely all the more upon the regularity of the beat and other indications. If they be not thus entirely lost, but are audible with greater or less distinctness, it must be borne in mind that, with an increase in bulk of *healthy* muscle, there is an increase in the contrast between the first and second sounds. The first sound is lengthened, and the second sound made sharper. As the

muscle becomes *unhealthy* or fails in power this contrast becomes less, and we may lay down the rule—"The more the first sound approximates in character to the second, the more do we fear that hypertrophy is being overtaken by dilatation or weakness." If the first sound is very weak, almost inaudible, this is, excluding fallacies of emphysematous lung, etc., a sure sign of failing muscle.

General Symptoms.—We can only repeat again that back pressure effects, if definitely present, are essentially signs of failure. Their absence or amelioration is provided for by hypertrophy, and hence they conclusively prove by their presence that the muscle is feeling the strain.

We may now, with these views of hypertrophy and dilatation, briefly examine the elements of the problem, "How is the heart itself affected by various valvular affections?"

Valves must be { Competent. / Incompetent. { Relative, from too wide an orifice. / Absolute, from intrinsic disease (thickened, puckered, etc.).

Orifices must be 1. Correctly adapted for their valves.
2. Narrowed (by adhesions).
3. Too large (by dilatation of chamber).

A moment's consideration of any single valve trouble will at once show that any pathological change in the structure or orifice leading to the leakage or stenosis must throw extra work on to that chamber which (reckoning from the direction of the blood stream) immediately precedes the lesion, and hence we might infer that hypertrophy should be the immediate result. But as the extra work is thrown on to the chamber at very different periods in its cycle of work and rest, the results are not quite the same in the two cases. In pure stenosis the extra strain comes during systole when the muscle is expecting to work, and here we can easily understand that almost pure hypertrophy results. In simple incompetency of a valve, however produced, the strain comes on the precedent chamber at a moment when the muscle is relaxing or resting, and hence, being caught unawares, so to speak, it is likely to yield somewhat, and an increase in the volume of the cavity is likely to ensue, or dilatation in its first sense, and, unless compensating energy or strength appears, dilatation in the second sense will soon appear. This precedent chamber (in incompetency at any rate) contains a larger quantity of blood than usual, which will, in systole, be forced into the succedent chamber at a time when it is relaxing

or resting, and hence in this chamber, too, there must occur some degree of dilatation to be again compensated by hypertrophy. When two or more valves are affected, and possibly in different ways, the problem becomes more difficult to follow, but the above principles will help us, provided we have the data as to time and amount of the several affections. Such data are, however, practically never offered to us, and we have to be content with accepting the general clinical result that in all cases of chronic valvular disease of the heart the organ becomes larger in its dimensions as ascertained by physical examination. This enlargement is nature's effort at compensation, and when the hypertrophy reaches its limits, or the muscle from any cause becomes badly nourished, cardiac failure and serious symptoms will supervene, whatever be the actual capacity of the chambers.

CARDIAC BRUITS, AND SOUNDS THAT MAY BE MISTAKEN FOR THEM

If, on auscultation of a chest, an abnormal sound be heard in the cardiac or aortic region, the first point to determine is, whether its origin lies in the cardiac or pulmonary organs. This, as a rule, is not difficult to decide. Pleuropericardial friction sounds resembling a pericardial rub, and compression of a small portion of lung tissue by the heart's movements, with a noise resembling a short systolic bruit, are the chief difficulties.

In the first place, they will either of them only be heard locally where they arise, somewhere along the margin of the heart. This and their rough character will alone be sufficient to separate them from endocardial bruits, which are heard over the bulk of the heart, and always conducted at least a little way from the point of maximum intensity. Then if by making the patient hold his breath for a few seconds the sound entirely ceases, we know at any rate that the *inside* of the pericardium is free from acute inflammation, and this is the most important clinical point, for an acute pleurisy here is only of additional importance from the liability to spread to the inner surface of the pericardium. If the sound does *not entirely disappear* on holding the breath, the heart movements must take some share in its production. Probably the pericardium is adherent to the heart on the one hand, and the pleura on the other. The position in which such sound is heard, on the boundary or just outside the limits of the heart, and a history of previous pericarditis or

pleurisy, will help us in deciding pleura *v.* pericardium, though frequently it is quite impossible to decide the point. Nor is the decision of very great importance practically, for either solution suggests adhesions which may in the long run interfere with the cardiac muscle, and are consequently to be feared in the future. It is well now to make the patient hold his breath—first at the end of deep inspiration, and secondly at the end of deep expiration. Should the abnormal sound be heard at one of these periods, and entirely disappear at the other, or, at least, be materially altered in character and loudness, it is probable that it depends on the forcible emptying of a small tongue of lung tissue by the mechanical pressure of the heart in systole. This is, however, a rare sound to hear.

If we have thus decided that the sound has its origin in the cardiac organs, the next point is to determine whether it is pericardial or endocardial. At the apex of the heart very little difficulty will be experienced, for the to-and-fro rubbing of pericarditis present throughout the whole cycle of the heart's action is very unlike any apical endocardial bruits, and the galloping rhythm of a rapid heart only requires a little careful listening to distinguish it. At the base, where pericardial bruits are perhaps more common, the difficulty of distinguishing them from double aortic murmurs is a little greater.

The following table correlates and contrasts the most important distinguishing features in all cases:—

	Exo- or Pericardial Sounds.	Endocardial Sounds.
Character.	Almost always a friction or rubbing sound; practically never musical.	More usually blowing in character; often musical.
Time of occurrence.	At any period of cycle, not specially with systole or diastole.	Always in connection with, if not actually at, systole or diastole.
Conduction.	Are not conducted along the blood stream or to apex, though they may be heard over a wide area.	Conducted upwards, or to the apex and axilla, except presystolic bruits, which have such special characteristics as to barely allow of mistakes.

	Exo- or Pericardial Sounds.	Endocardial Sounds.
Position of maximum intensity.	May be heard anywhere, but where heard do not gradually lose in intensity on gradual shifting of stethoscope.	Apex, for mitral bruits, base or mid sternum, for aortic bruits, are points of maximum intensity, and the bruits gradually diminish from their point of greatest distinctness.
Effects of pressure.	In yielding chests firm pressure is likely to cause marked alteration in sound.	Pressure makes no real difference in the sound, though the closer fit of the stethoscope may enable it to be heard more distinctly.
Variation from day to day.	Probably vary in area from day to day, and may disappear rather rapidly, but do not dodge about, appearing and disappearing alternately.	More likely to be constant, except in recent or ulcerative endocarditis; they have at times a curious habit of appearing and disappearing in a most capricious manner.
Pulse and associated symptoms.	Pulse rate sure to be accelerated in acute pericarditis (in chronic cases no rub because of adhesions, or if present is pleuropericardial, *vide supra*).	Pulse rate entirely dependent on the recentness of the inflammatory attack or on physical condition of patient, so that in chronic cases it may be for years quite natural.

Considerable precision may thus enter into our decision as to whether the source of an abnormal sound is endo- or exocardial, but when we come to consider the various problems connected with unusual sounds of endocardial origin, we are on very treacherous ground indeed. We can now and again decide some of them with a tolerable degree of accuracy, but on other occasions and for other problems even the keenest judgment of the most mature experience will only with diffidence and hesitation express an opinion.

Epitomised and tabulated the position is this:—

1. What is the exact origin of the bruit?

It may be due to:

- Blood states, poverty of blood, either of quantity or quality.
- Organic disease of valves obvious to the naked eye on post-mortem examination.
  - Mitral.
  - Aortic.
  - Pulmonary.
  - Tricuspid.
  - Insufficiency or stenosis, recent inflammation or effects of old trouble.
- Leakage of valves without naked eye changes. May be due to:
  - General dilatation of walls of cavities, or improper or feeble action of individual musculi papillares.
- Aneurysm.

2. What is the vital significance to the patient of the bruit thus detected?

Here I shall attempt only a complete solution of some of the simpler problems, with an indication of the principles that must guide us in attempting to decide the more complex ones.

A.—WHAT IS THE EXACT ORIGIN OF THE BRUIT?

(a) *Hæmic* v. *Organic Bruits*

It is usually comparatively easy to determine whether a bruit is due to poverty of the blood—ordinarily termed hæmic bruits—though the precise cause of such bruits is a matter of very great dispute and uncertainty; Dr. Byrom Bramwell offering us a choice of (1) pulmonary, (2) mitral, (3) left appendicular. Whatever its cause, the following particulars will suffice to separate it from bruits owning a definite organic lesion as causative factor:—

	If of Hæmic Origin.	If of Organic Origin.
Position.	Rarely heard at the apex, and if so, not conducted into axilla; usually heard at the base, and better a little to the left of the sternum.	Heard at apex, and often conducted into axilla; basic organic bruits are almost always better heard a little to the right of the sternum; pulmonary organic bruits being rare, except as congenital condition.

	If of Hæmic Origin.	If of Organic Origin.
Time.	Always systolic, never diastolic or presystolic.	May be, of course, systolic or diastolic, or both, or presystolic.
Character.	Not very distinctive, but they are never regularly musical.	May be musical.
Conduction.	Rarely conducted any distance beyond their point of maximum intensity; somewhat suddenly cease to be heard as stethoscope is moved.	Usually conducted some way from maximum point, and gradually die away in intensity as stethoscope is shifted.
Loudness altered.	Become very much louder on patient lying down on the back.	Not markedly altered by patient lying on the back; any alteration usually in direction of diminution.
Associated conditions.	Patient obviously anæmic, and as this improves, the bruit is likely to disappear.	Not necessarily anæmic; aortic disease is most likely to cause anæmia. This anæmia is not likely to yield to treatment, and if it does, the bruit still persists.

(b) *Valve of Origin of Bruits of Organic Origin*

The rules commonly laid down for discriminating the various sources of bruits due to organic valvular trouble may be briefly indicated as follows:—

Mitral systolic, indicating regurgitation through an incompetent mitral valve.	Systolic in time, *i.e.* accompanying or replacing the first sound; best heard at the apex, conducted into the axilla even to the angle of the left scapula; pulmonary second sound usually accentuated; apex beat displaced down and out in old standing cases, indicating some degree of enlargement of the left side the heart; pulse small.
Mitral presystolic, indicating abnormal narrowing of mitral orifice.	Presystolic in time, leading up to a sharply ceasing first sound; best heard a little above the apex, not conducted very far from its point of maximum intensity; pulmonary second

	sound very accentuated; apex beat if displaced at all, chiefly to the left—outwards—indicating enlargement of the right side. (If the word 'abrupt' be pronounced with a very rolling *r* the word becomes almost onomatopœic of the phenomena to be appreciated in connection with this bruit.)
Aortic systolic, indicating obstruction at aortic orifice.	Systolic in time, best heard over mid sternum about the third cartilage, or in the second right space; conducted upwards towards the vessels of the neck; apex beat displaced in old cases farther down and out than with any other bruit, indicating the greatest hypertrophy of the left ventricle; pulse small.
Aortic diastolic, indicating incompetent aortic valves.	Heard with or replacing the second sound, best over mid sternum opposite the third cartilages or in second space on the right; conducted down to the apex or to the ensiform cartilage, and so conspicuously as to be sometimes best heard at those spots; apex beat displaced down and out, again indicating a great degree of enlargement of the left ventricle; pulse peculiarly collapsing — the water-hammer or Corrigan's pulse.
Tricuspid systolic, indicating regurgitation through incompetent valves.	Systolic in time, best heard over the right ventricle, and conducted a little outwards to the right; very rare except in the later stages of heart failure, and is accompanied with a venous systolic pulse in the neck; indications of hypertrophied right ventricle pushing apex beat outwards.
Pulmonary systolic, or stenosis of pulmonary orifice.	Best heard to the left of the sternum, very rough; conducted upwards to the left.
Pulmonary diastolic; tricuspid presystolic.	Too rare to notice here.

In many cases, if used with discrimination, these rules will give tolerably accurate results, and are sufficiently useful to be remembered; but even in the most competent and careful hands they are apt to mislead, for statistics have shown[1] that "out of every hundred hearts we listen to, in thirty-six the bruits will give us exact information, in ten they will lead us absolutely astray, and

[1] Thesis for M.D. Oxon. based on some 700 autopsies.

in fifty-six they will give us information in excess or defect of the truth." The matter is of less importance, as we must iterate and reiterate the fact that every heart, with (or without) bruits, has to be judged on the merits of its muscle power and energy, and not by the bruits, the patient's condition must be taken as we find it, and not as one deducible from a theory of what it ought to be by the murmurs present. Of all factors by which our clinical judgment is influenced, that of the bruits present is the one of smallest weight.

The above rules will equally apply to bruits arising under circumstances which lead us to think that they are more likely due to relative incompetency or muscle imperfections, such, for instance, as a case in which cardiac failure is already noted, and a bruit becomes audible, unexpectedly developing under observation without pyrexia. The pathology of such cases will be touched on a little further in a subsequent section (*vide* pp. 135 *et seq.*).

(c) *The Bruits of Aneurysm* v. *Valvular Disease*

We have already (pp. 105 *et seq.*) dealt with the separation of aneurysms from intrathoracic solid tumours; we have here to separate them from lesions of the heart itself. Aneurysms of the transverse or descending aorta can scarcely give rise to suspicion of valvular mischief. The tumour and bruits and other local indications are too far removed from the situations where enlargement of the heart or valvular bruits would be observed. It is those of the ascending arch that require some care in discrimination, though even here the presence or absence of implication of the valves is of more importance than the presence or absence of an aneurysm as such.

The bruit of aneurysm is most frequently systolic, only occasionally double, practically never diastolic only, often also absent altogether; rarely conducted very far, and never down the sternum.	The bruit of valvular disease is most frequently double, occasionally systolic or diastolic only (very rarely found to have been completely absent when post-mortem shows gross valvular disease); usually conducted a good distance, and often heard down the sternum or at apex of heart.

Then the question of associated cardiac enlargement must be considered. My experience in the post-mortem room, in agree-

ment with recent observations, tends to show that aneurysms as such, *i.e.* when so situated as not to interfere with the aortic valves, have but little influence in causing hypertrophy of the heart. Aortic valve disease, on the other hand, is the especial trouble pre-eminently calculated to produce hypertrophy and dilatation of the left ventricle; hence, if with the bruit there is well-marked enlargement of the heart, the probabilities are strongly in favour of valvular disease; if there is but little or no evidence of such enlargement, the probabilities are in favour of an aneurysm as the cause of the murmur. Again, if there be a dulness extending suspiciously to the right of the sternum, the position of the heart's apex beat will give us help. Should this be displaced outwards much more than downwards, it is probable we have to deal with an enlarged right ventricle (lung trouble or mitral stenosis); if much downwards as well as outwards, aortic valve trouble is again probable. If not very materially displaced, aneurysm again is strongly suggested (or solid tumour).

It has to be admitted that the general conditions, viz. severe physical exertion over long periods, alcoholic excess, syphilis, advanced age with senile degeneration, renal disease, etc., which are predisposing factors in aneurysm, are equally the predisposing factors in primary aortic valve disease (the valves are, in fact, merely a part of the aorta, and degeneration with aneurysmal yielding may occur at any point), so that it is by no means an unfrequent thing, in fact I think it is a rule of the majority of cases, that when aneurysm occurs it shall be associated with valvular lesions, and hence it is frequently impossible to exclude the aneurysm, although we have positive evidence that the heart is affected. The diagnosis is of less importance in that the condition is a grave one, and what is likely to help the heart is likely to help the patient to bear with, or to cure the aneurysm if it exists.

B.—WHAT IS THE VITAL SIGNIFICANCE TO THE PATIENT OF THE BRUITS?

This includes two problems, the first of which, viz. the bruit as an indication of the condition of the muscle of the heart, has really more to do with prognosis than diagnosis; it has already been touched upon in dealing with the symptoms that suggest morbus cordis, and will be further considered (*vide* pp. 135 *et seq.*); the second is the bruit as a direct indicator of leakage or stenosis, and the

influence of these on the patient's condition; this we will now consider.

Bruits of hæmic origin may certainly be ignored in cardiac pathology. They are accidental phenomena in diseases of other origin, and disappear with the disappearance of the blood dyscrasia, and their significance is nil beyond the significance of this dyscrasia.

Bruits of aneurysmal origin, too, have the significance of the aneurysm itself. They add nothing to our knowledge of the course the arterial disease will take; they give no guidance in treatment, and their disappearance, if they do disappear, does not necessarily mean the cure or amelioration of the condition. This work does not deal with treatment, but I cannot refrain from putting on record an emphatic protest against any *extremes of active treatment* in aortic aneurysms, such as starvation and severe bleedings. The lesion is one that will kill quite fast enough without our assistance, and our efforts had better be directed to making life bearable by counsels of moderation in all things than to rendering miserable the remaining years of a threatened life.

Bruits of undoubtedly organic valvular origin have the following significations:—

(1) To determine the presence of recent inflammation (*vide* p. 116).

(2) To determine excessive dilatation of a chamber (*vide* p. 122).

(3) By their disappearance to determine muscular weakness (*vide* p. 140).

(4) To determine single or combined alterations of old standing in the valves, with their significance.

This last (4) is the problem to the decision of which I devoted some time ago a very considerable amount of statistical inquiry. Omitting the actual figures, the tables I prepared showed that, supposing the patient died from the effects of heart disease—

(*a*) Mitral incompetency gives, amongst single lesions, the worst prospect of prolonged life.

(*b*) Aortic incompetency also amongst single lesions gives the best prospect.

(*c*) The question between aortic stenosis and mitral stenosis depends on other factors to such an extent that the

isolated lesion is of very small importance, and probably the prospects are about equal.

(*d*) If aortic stenosis be combined with mitral incompetency, the patient gains about three and a half years of life from what he would enjoy with aortic stenosis only.

(*e*) Mitral stenosis combined with aortic stenosis is a very fatal combination; but if the pulmonary or tricuspid be also stenosed, the patient gains about seven years of life. Such a marked difference points strongly to the lung as the weak spot in heart disease.

These results, at any rate the first one (which seemed to be the most firmly established by figures), are so startling and so contrary to what is apparently one's general experience of the innocuousness of a mitral systolic bruit, that I was then (1890), and still am, driven to the conclusion that they illustrate more forcibly than any other argument the futility of attempting to lay any weight on the nature of the bruits in estimating the value or power of a heart whose valvular machinery is damaged. I leave them, without further comment, to be refuted or corroborated by future investigators.

HEART DISEASE IRRESPECTIVE OF MURMURS

In discussing hitherto the pathology of cardiac disease and hypertrophy and dilatation, we have had in mind chiefly chronic organic disease of the valves as the essential cause of the disturbance. It is certain that the nervous and muscular mechanisms, connections, and structures are capable, in an otherwise fairly healthy individual, of obviating to an almost complete extent, and for a very long time, such defections in valve arrangements; but if the patient is to die, as many do (though very many do not) from chronic valvular trouble, a time eventually arrives for him when such compensation fails from malnutrition or overstrain of muscle (? of nerve too). The case then, from its causation, goes into a special group, but from its clinical features remains an illustration of the class of cases which we must now consider, viz. those in which the nervous or muscular mechanisms have broken down, or possibly (as in valvular trouble?) both have given out. The precise pathology of the various examples of this class is often most obscure, and probably the essential elements in it vary from time to time, now nerve, now muscle being at fault. So far as can be understood,

I think the following table represents fairly completely the causes which may be at work in producing the clinical features of the case:—

Muscular defects of degeneration (whether fatty or fibrous or other is not very material; the muscle is no longer natural).	Of slower onset through months and years.	Malnutrition from the circulatory disturbances in the heart itself, of valvular disease; common.
		Malnutrition from more general conditions, *e.g.* age, vascular degeneration, kidney disease, gout, obesity, etc.; common.
		Definite new growths, syphilis, tubercle, malignant disease, hydatids, etc.; all are rare.
		Fibrosis or scar formation from any cause, especially peri- or endocarditis, syphilis, etc.; also common, at any rate as revealed by the microscope.
	Of more rapid onset, possibly days or weeks.	Acute febrile disease, especially perhaps pneumonia, typhoid, and septicæmia (from pyrexia, or toxins, or both). Poisons, such as tobacco, alcohol, etc., which may affect the muscle as well as the nerves. (This group may come on more insidiously.)
Nervous influences or defects.	Purely reflex.	From diseases of any organ, *e.g.* intestines, stomach, ovary, etc. (? thyroid).
	Direct effects on vagi or accelerators.	Pressure on peripheral part of nerves, enlarged glands, *e.g.* central pressure of tumours, meningitis, etc.
		Loss of control of nerves from actual neuritis or smaller degree of nerve illness.
		Poisons—tobacco, alcohol, probably thyroid secretion, etc. Diphtheria, influenza, and other specific diseases probably should also be placed here.

The clinical features of the cases are as varied as the possible causations. Sometimes they assume the shape of the most dangerous and serious illnesses we can meet with; sometimes, on the other hand, they indicate but a trivial malady; yet the former may prove of no moment, while sudden death may terminate the latter. They may present all the general indications of back pressure, or they may confine themselves almost entirely to local pain and distress. Diagnosis becomes increasingly difficult with the absence of indubitable signs of organic disease, and mistakes must constantly occur even to the most experienced. Still, a careful inquiry into the history of the case, and an equally careful weighing and balancing of all available factors must be undertaken, and will help us materially in, at any rate, basing our opinion on a sound foundation of probability, beyond which clinical medicine can rarely go; certainty is rarely attainable where the exceeding delicacy of ill-understood physiological balances is in question.

It may be laid down as a general rule (not perhaps without some very grave exceptions—vagus neuritis, for example, after diphtheria) that affections of the nerve apparatus are less serious in their outlook than those of the muscle. Remembering how easily the heart beat is affected, at least in frequency, by every passing mental shock, by pain, by gastric disorder, by, in short, reflex influences from every quarter, the general truth of this rule in practice must, I think, be accepted. Hence our first efforts at diagnosis should be directed to an endeavour to determine on which side weakness lies. The following paragraphs in parallel columns will show the points of distinction more clearly, and bring them into sharper contrast.

DERANGEMENTS OF THE NERVOUS CONTROL	DERANGEMENTS IN THE MUSCLE APPARATUS
Points in the Previous History	
If previous attacks of symptoms have occurred, they have probably cleared up entirely, leaving no trace of permanent and persistent signs of ill-health. They were not associated with decided evidence of back pressure effects.	Previous attacks of symptom have probably not cleared up entirely, but have left permanent shortness of breath or other evidence of ill-health. They were probably associated with definite signs of back pressure. In

The first attack was probably associated with some mental shock or dyspeptic trouble, or if with a definite illness, it was more likely influenza or diphtheria (both known to cause nerve changes) than rheumatism or scarlet fever, etc. (known to produce endo- or myocarditis). Attack more likely to be quite sudden in onset, without obvious exciting cause.

young people the first attack traceable to rheumatism or other disease known to produce organic changes in endo- or myocardium. In elderly patients there will probably be found an admission of progressive feebleness and shortness of breath previous to an actual attack of symptoms calling for medical aid.

Points in the Symptoms Complained of

More likely to be stress laid on the local symptoms, pain more or less sudden and spasmodic in the cardiac area (rapidly subsiding after eructations or vomiting), palpitation, fluttering sensations, choking, etc., also prominent, but soon subsiding. Likely enough to return on a repetition of the recognised cause, *e.g.* heavy meal, tobacco, alcohol, etc. Faintness is again a very common phenomenon in such cases, but fatal syncope extremely rare.

Local symptoms less prominent, and usually masked by the general distress. If they are prominent there is no doubt about their genuine severity (angina, *vide* p. 113), and they do not subside suddenly after mere eructation or vomiting. Exciting cause much more frequently physical exertion of some kind, or very violent excitement. Faintness of an ordinary character is not common, but fatal syncope is fairly frequent.

Points ascertained by Examination of the Patient

General Considerations and Conditions

Age.—In the young the two great causes of cardiac *muscle* weakness are valvular trouble (congenital or acquired) and acute pyrexial disease. If these two can be excluded, cardiac complaints in younger subjects are almost certainly nervous in origin. In older patients, when their complaints may point to a cardiac source, we are much more anxious about the condition of the muscle. This anxiety will be increased very much by evidence of renal disease or arterial degeneration. The latter, in fact, is a better gauge of age than the calendar's tale of years. The coronary

arteries so frequently share in this degeneration that a degenerate radial or temporal is an *absolute bar to our regarding lightly any history of cardiac attacks;* they compel a serious view being taken of such a case. Fatal angina or an aneurysm, otherwise undiscoverable but with a gloomy outlook, must inevitably occur to our minds under such circumstances. In later life, too, one is inclined to look more favourably on the same symptoms in a woman than in a man.

Sex.—Of sex, apart from age, but little can be said. Women, as a rule, are certainly more liable than men to view with apprehension slight symptoms or disquieting sensations in the cardiac region; hence, if there is no obvious origin for heart disease, one would be more inclined in a woman towards a neurotic explanation of a case. If, on the other hand, obvious hysterical manifestations of other kinds are present—Grave's disease, influenza, tobacco, alcohol, sexual indulgence, masturbation, puberty with its strain on the system, a neurotic heredity—if any one of these gives us a clue, sex at once becomes of very little account in comparison with such indication.

Back Pressure Effects.—These we have already (p. 118) considered in some detail. For our present purposes we may divide them into —(1) disturbances of function; (2) disturbances of structure—œdema, fibrosis, etc.—and we may state that—

In nervous affections of the heart the latter class is seldom (? never — vagus neuritis, for example) seen. Evidence of the former class is fairly common: shortness of breath without rales or bloody expectoration, disturbances of general cerebration, or of special senses, or irritability of stomach, etc.	In muscular troubles both classes are common, and, indeed, the characteristic feature. Provided there is no local cause for a member of the second class, its occurrence is practically pathognomonic of heart muscle failure.

Local Examination of the Cardiac Region

Inspection, *palpation*, and *percussion* are only of essential utility in determining the presence or absence of hypertrophy and dilatation, and as one indication of frequency and irregularity.

Hypertrophy and Dilatation, either or both

In purely nervous affections of the heart the organ is but rarely	In muscular troubles alteration in the size of the heart is constant

much larger than normal. The chief exception is long-standing Grave's disease.

except in acute febrile disorders (on the presence of which the diagnosis mainly rests). This, in fact, when marked, constitutes the type case.

Frequency and Irregularity

Tachycardia and bradycardia are the commonest features in nervous affections of the heart. In fact, either of them, possibly with some irregularity, but *without other objective signs of heart disease*, constitutes the type case, and under such circumstances, in combination, a pulse of over 160 or under 50 may safely be regarded as due to nervous influences.

In the more slowly developing defects of muscle, frequency rarely rises much beyond 130 without at the same time being associated with marked tumultuous irregularity and signs of back pressure; nor, on the other hand, is it often reduced below 60 without similar indications. In the more rapidly developing muscle failure of poisons and pyrexia frequency may be very high without such other symptoms, but then the feebleness of the sounds is our best guide.

Tobacco in excess, Grave's disease, influenza are the common causes of such tachycardia and palpitation, and careful inquiry must be made for them.

Irregularity alone as the *sole indication* of muscular trouble never occurs, at any rate for long. Should it be the first warning others will very soon appear.

Vide frequency also (on p. 124) under Hypertrophy and Dilatation.

Bruits.—In nervous troubles bruits of any kind are exceedingly rare, and still rarer is it to find one that follows the rules laid down for those of valvular origin, though I think it probable that some of the obscurer murmurs may be due to irregularity of nerve influence on individual muscle bundles.

The presence of bruits in muscle troubles has already been discussed (p. 116). Here we need only mention that the disappearance of a valvular bruit *with* increase in severity of symptoms is pathognomonic of muscle failure, as, indeed, is its first appearance under such circumstances of increase in symptoms.

The Ordinary Cardiac Sounds

Nervous affections of the heart have a peculiar tendency to render the sounds sharper and more distinct as noises, though the contrast between the first and second may be lost in extreme frequencies. If any sound disappears in nerve troubles it is the second.

Muscle troubles, on the other hand, have a tendency to shorten the first sound, and, at the same time, to render it less audible. So much so, that in some cases of poisoning, prolonged pyrexia (as in typhoid, etc.) or fatty heart, the first sound may become almost inaudible. This is true whether bruits are present or not. Their presence only renders its appreciation more difficult.

Such, in detail, are the individual points requiring attention in the diagnosis of the nature and meaning of cardiac complaints. In practice the difficulty lies not so much in estimating these separately as in weighing them collectively and striking a balance between those which point in opposite directions. It is here that clinical experience and acumen have their most brilliant opportunities for making a reputation for their possessor, and no hard and fast rules can replace them. The only advice I have to give of general application is, "Don't be in too great a hurry to express a decided opinion. Time and circumstances will often throw a clear light on a case which is at first apparently obscure and perplexing."

ULCERATIVE ENDOCARDITIS

To understand the diagnostic problems of ulcerative endocarditis it is necessary to enter into the outlines of the pathology of endocarditis in general. The four following postulates constitute the essential foundations:—

1. That acute endocarditis arises, precisely like inflammation elsewhere, as the result of an irritant applied to the membrane.

2. That the severity of the anatomical lesions, produced by the inflammation, is strictly proportionate to the relative strength of—on the one side, the toxicity or nature of the irritant; on the other side, the resisting power of the membrane.

3. That in clinical practice the irritant is always either chemical

substances produced by (1) unnatural metabolism of the tissues,[1] (2) microbic activity, or it is the microbes themselves and their activities locally exerted on the membrane.

4. That a previously damaged valve forms a *locus minoris resistentiæ* to the influence of any poison circulating in the blood.

In connection with postulate (2) and our knowledge of the anatomical changes that ordinarily accompany any inflammatory process, we may tabulate endocardial changes thus:—

Phenomena of constant occurrence at the onset in every case of acute endocarditis.	Phenomena whose occurrence depends on circumstances defined by postulate (2).
Active congestion, with effusion of serum and cells and swelling of the valve.	(*a*) Simple swelling without loss of epithelium, no vegetations, and ultimately complete resolution.
	(*b*) Swelling plus loss of epithelium (microscopic ulceration); appearance of vegetations; ultimately some thickening or scarring left behind.
	(*c*) Swelling, loss of epithelium, vegetations plus loss of other tissues and naked eye ulceration.

(*a*) Must be allowed to occur in those cases of rheumatic fever and other illnesses in which a definite valvular bruit occurs, but finally entirely disappears.

(*b*) Is the most common clinical occurrence in such illnesses (rheumatic fever) as leave a permanent organic valvular bruit. These must in strictness be also termed ulcerative endocarditis, but as the loss of tissue is of microscopic proportions, the term is usually reserved for the third class.

(*c*) It is to this condition that the term ulcerative endocarditis is usually applied. I have put it in serial continuity to the other forms to emphasise what I believe to be its real position, viz. an endocarditis, the severity and course of which depend upon circumstances which may be called accidental, in support or explanation of which statement we will now consider the clinical occurrence of severe endocarditis.

First of all, cases of the following type are unfortunately by no means rare. A patient, probably under twenty, is attacked with

[1] This is inserted in view of the doubt whether rheumatism is a microbic disease.

rheumatic fever. A valvular bruit develops and persists, but the patient becomes more or less convalescent from distinct rheumatic symptoms. Then, without any pyrexia, or very slight and temporary fever, one or other, or perhaps one after the other, some of the following incidents occur: attacks of pain in and swelling of the spleen, renal pain with hæmaturia, cough with bloody sputum and a pleuritic rub, œdema with loss of circulation in a limb or part of one, hemiplegia, etc., each and all evidences of an embolus being swept from the heart into the organ specified. The patient may yet live on in an invalid condition for some months, and even for years, and eventually succumb from cardiac failure or from an embolus proving fatal. On autopsy we find the areas of embolism, corresponding to the symptoms, each and all in what may be termed a quiescent condition, *i.e.* one the ultimate end of which will be simple fibrosis or scarring after absorption of very slightly irritating dead tissue. We find on the valves of the heart a greater or less amount of vegetations, on the removal of which losses of substance—ulcers—may be plainly visible in the valve tissue, but, and this is the point, *from neither the valves nor the embolised areas can any growth of pathogenic microbes be obtained.* Such a case must be termed severe—and ulcerative—endocarditis, though its origin is purely rheumatic.

Now let us sketch the other form. An attack of rheumatic fever, followed by the appearance of a bruit as before, but the patient gets quite well of all symptoms and remains so, a murmur being the only evidence of his attack. One day an entrance to his system is effected by potent-for-evil microbes, and now follows a train of symptoms which in gross may be termed septicæmic—hectic, sweating, rigors, diarrhœa, ups and downs—and quite probably evidences of emboli, as in the previous case. The patient dies, not of an apyrexial exhaustion or cardiac failure, but evidently of some acute febrile process. On autopsy we find, not quiescent, absorbing embolised areas, but the seat of each embolus is occupied by a soft, semi-purulent collection of debris, the heart valves or endocardium may be in precisely the same condition *to naked eye* as in the previous case—though it is probable that the loss of tissue will be more evident—vegetations may or may not be equally abundant, but *from the ulcer and from each embolised area can be obtained a culture of pathogenic microbes.* Microbes of many kinds have thus been found—gonococci, pneumococci, pyogenes albus, p. aureus, etc.

The difference between the two cases is clinically real enough

and serious enough, though the anatomical changes to naked eye may be practically identical, at least in the heart. But the difference is, I maintain, a quasi-accidental one, and due to a contamination of the blood by virulent microbes, which (*vide* above) may be of almost any kind, and hence ulcerative endocarditis is not a specific disease to the same degree as are scarlet fever, syphilis, or small-pox, for example. It is more like pneumonia, which can be produced also by many microbes.

Diagnosis of Ulcerative Endocarditis

Septic or malignant or ulcerative (they are all used synonymously in clinical medicine) endocarditis is either endocarditis plus blood-poisoning, or it is blood-poisoning plus endocarditis, according as the valves have been previously or are now only for the first time damaged. Its three diagnostic features are: (1) symptoms of blood-poisoning; (2) embolic phenomena; (3) valvular bruits—and we have to see how it is to be separated from the diseases which may at times present these features.

Simple endocarditis, by which we must here understand one from which recovery, except as regards scarring of the valves, is easily possible and indeed the rule, has been sketched above, and the principal point of difference between it and the malignant form is the absence of those extreme variations in temperature, of diarrhœa, and of rigors, which indicate that blood-poisoning has been superadded to the valvular trouble, though the emboli and the murmurs may be common to both.

To diagnose septic endocarditis from an ordinary case of septicæmia, no matter what was the point of entrance of the poison, is a work of supererogation; they are identical. The only point is, that the presence of a bruit renders it probable that the enemy has effected a lodgment in a position—the cardiac valves—whence his spread over the country is supremely easy. If the bruit materially and distinctly alters from day to day, we have strong proof that active anatomical changes are going on in the valve—destruction of tissue or disposition of fibrinous vegetations on a raw surface—and these make the diagnosis nearly certain.

But there are still three specific and special causes of toxæmia which are likely to cause confusion. These are typhoid fever, acute tuberculosis, and ague.

Typhoid fever, in its pyrexia and in its diarrhœa, may resemble, and is, a septic disease; *per contra*, a person with old valvular

trouble is not thereby protected from enteric, and in such a person the likeness to septic endocarditis may be very close. The following differences will, however, usually help us: (*a*) the fever in typhoid is usually more regularly persistent, not so intermittent or remittent in its type; (*b*) the abdominal tenderness, the headache, and the rash are none of them common in ulcerative endocarditis; and lastly, (*c*) the serum (Weidal's test) test for typhoid will, with almost mathematical precision, tell us whether typhoid is or is not present; moreover, (*d*) if a bruit is present in a patient with typhoid alone, it will not alter from day to day in any marked degree. With reference to this last point, I must add that, in my view of the pathology of septic endocarditis, it would certainly be possible for typhoid to relight ulcerative changes in the valves.

The diagnosis from acute tuberculosis must mainly rest on the positive evidence of this disease being present—a preponderance of pulmonary signs and symptoms, proof of other tubercular lesions, etc. If a cardiac bruit be present, it must be watched for the same indications as when typhoid is suspected. Too often, it must be admitted, the diagnosis is only cleared up by post-mortem examination.

Malignant endocarditis has been mistaken for ague only by reason of a temporary regularity in the rigors and pyrexial extremes. The effects of quinine and a few days' delay have never left the matter long in doubt, unless death has too rapidly intervened.

Many cases have now been reported in which, while cardiac bruits have been absent during life, autopsy has revealed the presence of (septic?) valvular lesions. Of such cases it can only be said that, without the assistance of a combination of septic symptoms and embolic phenomena, diagnosis is simply impossible. We can only guess at it by an attempted process of exclusion.

Prognosis in Heart Disease

This involves two separate lines of thought. First, the immediate prognosis (*a*) in a case of acute endocarditis, (*b*) in a case of cardiac failure without evidence of acute inflammatory changes; secondly, the general prognosis of a case of chronic cardiac trouble as regards occupation, etc.

In a given case of acute endocarditis the prognosis depends entirely upon the severity of the inflammatory process, and the extent to which it weakens the muscle by its spread. The deter-

mination of this is entirely beyond our powers of direct observation; we can only indirectly estimate it to some degree by the symptoms of cardiac failure, and prognosis can only be made from day to day by noting each day's progress. When the temperature (rarely much raised) becomes quite normal we have a right to assume that the active stages of inflammation are at least not progressing, and then intensification or amelioration of back pressure symptoms gives us a daily despair or hope, always with a tendency to look on the bright side, because, as a matter of clinical experience, it is but rarely that acute—provided that it is not septic—endocarditis kills directly. Recovery is the rule, at least to the extent of a capability of leading a life which may be useful, and even enjoyable. When, as in repeated attacks of acute rheumatism, we have reason to believe that fresh outbursts of inflammation keep on recurring, the outlook must necessarily become more gloomy with each attack, though even here we may, to a large extent, accept the above suggestions.

In considering those cases in which we do not suspect recent endocarditis, whether the primary trouble has originally been valvulitis or is now essentially a primary muscular degeneration, the most important question is, What is it that has caused this heart to fail *now*; is it that the work has been too great or the power too small?—for evidently the ratio between them is one which cannot be maintained. Careful inquiry must be made into the duration and progress of each symptom, however trivial, and into the habits and surroundings of the patient; and the less obvious appears the excess of work—we must never forget that worry or anxiety, mental work, in fact, may be equally or more detrimental than bodily exertion—the more anxious are we lest it be power that is failing below a standard compatible with prolonged life. When we have duly weighed the items of this consideration, we can only await next the verdict of treatment. Statistics of bruits, with ages at death, are but of little use. As age or degeneration, *i.e.* loss of vital elasticity, advances we must naturally expect a slower response to remedial measures, if, indeed, response be not entirely absent; but even now it is astonishing to see how cases may improve by a judicious combination and alternation of drugs and of rest and increasing exertion. The latter, though an obvious common-sense method of treatment, has recently been brought into fashionable prominence under the name of Schott or Nauheim treatment.

The prognosis of other cardiac affections, though requiring frequently the nicest of judgment, can generally be regarded with

lenient eyes. In nervous affections, as a group, the outlook is certainly good, and the patient must be encouraged to live an ordinary life, with a few hints as to the avoidance of excesses of mental or bodily strain; and if a cause, such as tobacco or alcohol, can be discovered, moderation or total prohibition must be enforced. Even when associated with other phenomena of Grave's disease, a rapid heart is comparatively harmless, though a prolonged period of ill-health must be accepted as inevitable.

When your advice is asked by a patient who has recognised valvular disease, with no present symptoms of failure, we must look, and encourage the patient to look, on the cheerful side as much as possible. Nothing can be more injudicious in such cases than to pull a long face and condemn such a patient to a life of idleness, ticketed with a label, "Heart Disease." Habits of depressing introspection are sure to arise, and it is these that kill more surely and rapidly than any valvular lesion. Pregnancy is a serious consideration, but I have known a girl of seventeen with severe mitral trouble of rheumatic origin marry at twenty, bear eight healthy children, and survive till the age of seventy-seven. In questions where a possible pregnancy has to be considered, I think a past history of back pressure symptoms of more importance than the present character of a bruit.

CHAPTER V

DIFFERENTIAL DIAGNOSIS OF SOME SYMPTOMS AND AFFECTIONS OF NOSE, THROAT, AND ALIMENTARY TRACT AND ANNEXA

HÆMORRHAGE

HÆMORRHAGE may *appear* at the nose, mouth, or anus as the three natural openings of the system; but it may *originate* from many situations, and we shall try to indicate the reasons which lead us to assign to it its actual seat of origin.

EPISTAXIS, OR BLEEDING FROM THE NOSE

The nose differs very materially from the mouth and anus from our present point of view, because there is practically no difficulty in ascertaining that the blood does come from somewhere in the naso-pharyngeal cavity itself. When blood makes its exit from either of the other two openings the question of precise origin is very much more intricate, as we shall presently see. Only as the very rarest of clinico-pathological curiosities could one imagine a gastric or pulmonary hæmorrhage giving rise to more than a passing suspicion that the naso-pharynx was its seat, and very little inquiry into the mode of onset, and physical examination of the cavities, will be required to rapidly settle that suspicion should it have momentarily arisen. Consequently, in a case of epistaxis of some duration and amount, the only primary questions of diagnosis that can arise are (1) its precise seat, and (2) its cause.

The determination of question (1), though often difficult, or even impossible, is, if it be necessary at all, entirely a matter of local inspection with mirror and speculum, and will not be further

considered. The answer to question (2) is a matter of the very widest clinical importance, and we will proceed to classify the—

Causes of Epistaxis

	Diagnostic Points.
1. Local Causes: in Nose, etc.—	
(*a*) Traumatism, fractured base of skull, blows, etc.	History usually obvious; difficulties only arise when patient is drunk, or otherwise *non compos mentis* or unconscious.
(*b*) Adenoids.	Subject a young child; suspected by nasal voice, sunken bridge of nose, habit of mouth breathing; proved by inspection with finger and mirror.
(*c*) Malignant growth or innocent polypi.	Subject adult probably; may be complaint of local pain, and of nose being stuffed; inspection clears up the case.
(*d*) Ulcers, lupus, syphilis, etc., and necrosis.	Detected by speculum; very probably associated with ozæna.
(*e*) Diphtheria and similar acute local trouble, or even simple catarrh.	History of illness preceding the epistaxis, and probably other local signs, swelling, etc.
2. General Constitutional Causes—	
(*a*) Hæmophilia.	Invariably (in first attack) a young subject, most likely a boy; history of previous attacks of hæmorrhage markedly disproportionate to the lesion, *e.g.* tooth extraction, etc.; family history sure to give evidence of heredity.
(*b*) Leucocythæmia.	Probably a young subject; examination of spleen and blood will clear up the case.
(*c*) Vascular degeneration.	Subject old (physiologically, if not in years); radials thickened, heart hypertrophied; possibly also renal symptoms.
(*d*) Onset of acute fevers.	Subject young; obviously out of health; thermometer proves pyrexia.
(*e*) Scurvy.	Swollen and spongy gums.
(*f*) Purpura.	Always (if epistaxis) purpuric spots elsewhere.
(*g*) Puberty.	Age the most important guide, coupled with the knowledge that epistaxis does occur frequently at this epoch without other obvious causation.

2. General Constitutional causes—	Diagnostic Points.
(*h*) Vicarious menstruation (?).	Obviously a female, and with a history of suppressed natural menstruation, but the occurrence of well-established cases is very doubtful; the bleeding would be periodical.

These causes, as well as those which follow, emphasise the absolute necessity of a careful systematic examination of a patient with epistaxis if the diagnosis is not at once obvious.

3. Local Conditions other than Nasal—	Diagnostic Points.
(*a*) Distinct kidney disease.	This is only a slightly special branch of 2 (*c*) in which the kidney trouble is, and has been, a prominent feature in the case. It is not absolutely essential that contracted kidney should be present. A consecutive nephritis (*q.v.*) will sometimes cause it.
(*b*) Cirrhosis of liver.	History or other strong indication of alcoholic excesses; liver probably enlarged.
(*c*) Morbus cordis.	Bruits present, or at least other evidence of cardiac disease.
(*d*) Pregnancy.	An occasional cause of epistaxis. History and abdominal examination sufficient.
(*e*) Coughing, sneezing, etc., to excess.	History distinctive; onset after such excessive respiratory movements.

Differential Diagnosis of Blood issuing by the Mouth

Blood which issues by the mouth may have obviously many sources:—

1. Naso-pharynx.
2. Mouth and pharynx.
3. Stomach or œsophagus.
4. Lungs.
5. Aneurysms bursting into either viscus.

The history of the way in which the blood comes up for evacuation may in some cases be sufficient and diagnostic, but even in intelligent adults the question of vomiting *v.* coughing is often very difficult to settle, so that the differential diagnosis must proceed on definite exclusion lines by objective guides as far as possible.

Our first care, then, must be, in a doubtful case, to carefully wipe away the blood from nose and mouth and fauces; a careful local examination, with thorough exploration with eye or finger, will then soon show us whether the blood is coming from above, and reference to the causes of epistaxis will probably clear the matter up after a systematic examination of the patient.

N.B.—Do not be led away from a careful examination of nose and throat by a definite history of hæmatemesis (*q.v.*) or hæmoptysis (*q.v.*), especially if other symptoms pointing to stomach or lungs are absent.

Suppose, now, that local examination reveals the mouth or fauces as the seat of the bleeding vessel—the following conditions must be borne in mind as further elements in the exact diagnosis:—

Causes of Hæmorrhage from Mouth and Fauces

	Diagnostic Points.
1. Traumatism from external violence.	History obvious.
2. Traumatism from teeth or food.	Wound found on gums, tongue, cheek, or fauces; its history must be most carefully inquired into, whether consciously done by the teeth or during a meal, or attack of coughing, or whether the patient is unconscious of its origin. *A bitten tongue with no history, and with blood on the pillow in the morning, in a youngish subject is* prima facie *strong presumptive evidence of nocturnal epilepsy.*
3. Gums.	Spongy, from scurvy or purpura or anæmia, all of which will show definite features elsewhere. Gingivitis in dentition, or from want of cleanliness in mouth, easily recognised by age, or by tartar encrustation.
4. Ulcers.	All fairly easily recognised—malignant, syphilitic, lupus, tubercle, or simple or gangrenous (cancrum oris).
5. Acute tonsillitis and pharyngitis.	Easily recognised by dark purplish red colouration, or by actual ulceration.
6. Chronic pharyngeal conditions.	Blood only in spots; dilated and varicose veins seen on posterior pharyngeal wall.

As sources of hæmorrhage of a serious degree none of these

require much consideration beyond mention of the fact that any deep or progressive ulceration may open a large vessel; but as sources of alarm to a patient they have a very great and real importance, and a discovery of one of them may go a long way in establishing a reputation for care or carelessness.

Leaving the mouth and parts open to inspection, we have now to consider those seats of a hæmorrhage appearing through the mouth, which are out of sight. These are—

1. The air passages,
2. The alimentary tract,

and our first point in diagnosis is to determine which of these two main tracts is at fault, in other words, to distinguish—

	HÆMOPTYSIS	*v.* HÆMATEMESIS
Sensation accompanying the voidance of the blood.	Cough or tickling in throat, or possibly no warning at all.	Nausea first, followed by act of vomiting
Precedent condition of patient.	Cough or known lung mischief.	Dyspepsia or marked gastric disturbance.
Associated condition of patient.	If pallor present, it came on subsequently to the hæmorrhage, *i.e.* the bleeding excites cough before it is sufficient to cause pallor.	Pallor often precedes the ejection of blood, *i.e.* blood pouring into stomach causes it before the blood is voided.
Voided blood itself.	Usually frothy, and often bright (mixture c̄ air), always alkaline.	Never frothy, usually dark, and frequently acid from admixture of gastric juice.
Associated phenomena.	Melæna not marked as a rule; some physical signs, rales, etc., possibly found in lungs. *N.B.*— THEIR ABSENCE DOES NOT EXCLUDE HÆMOPTYSIS.	Almost certainly followed by melæna; possibly local pain or tenderness over abdomen or stomach. *Caution* — muscles may be tender from coughing or straining.

Having satisfied ourselves that it is (1) the lung which is the source of the blood, diagnosis must next proceed to the—

Causes of Hæmoptysis

	Diagnostic Points.
Laryngitis, tracheites, bronchitis acute, and granular pharyngitis.	If within reach of the laryngoscopic mirror, cause soon cleared up (tubercular, syphilitic, or malignant ulceration, *q.v.*, all of them common causes of slight hæmoptysis); blood either in little pellets or thin streaks; sputum very sticky and frothy; pain locally on swallowing or coughing.
Pneumonia.	Sputum usually *uniformly* stained either slightly or up to prune-juice colour, very sticky and usually nearly or quite airless; associated dulness, tubular breathing, and temperature, etc., commonly distinctive enough.
Phthisis.	Far and away the commonest cause of profuse hæmoptysis; bacilli if found distinctive, but diagnosis in early stages often impossible.
Gangrene, abscess bronchiectasis.	All rare causes of hæmorrhage; associated fœtid smell of sputum, and local physical signs characteristic.
Degenerative bronchitis and emphysema.	Condition described by Sir Andrew Clark: hæmorrhage fairly free, physical signs of bronchitis and emphysema, *without* any distinctive bacilli or evidence of excavation or dilated tubes.
Foreign body.	History usually sufficient of choking with aspiration of the foreign body.
Aneurysm.	As a cause of hæmoptysis rare, but when aneurysm is present hæmoptysis is tolerably frequent; if other signs of aneurysm present, hæmoptysis adds to their weight, and especially if there are signs of pressure on a large air tube. *N.B.*—Slight hæmoptysis may precede for a long period the actual rupture of the sac.
Malignant growths.	Said to be like red-currant jelly, but in my experience the hæmorrhage here is only distinguishable by the physical signs of the growth, and has nothing distinctive in itself.
Blood aspirated into lung in hæmatemesis.	*Vide* tests between hæmatemesis and hæmoptysis; if definite hæmatemesis proved to be present there will only be slight pellets of blood or blood-stained mucus coughed up for a few hours or a day or so after the vomiting.

These causes really fall into two clinical groups, viz. those in which the source and cause of the hæmorrhage are obvious or

easily detected, and those in which probabilities have to be nicely balanced. To the former group belong visible laryngeal and pharyngeal troubles, obvious gangrene, abscess, aneurysm, pneumonia, and carcinoma secondary to an obvious primary growth. To the latter belong those cases of definite hæmoptysis in which no physical signs at all are to be found, and those in which the physical signs are those of bronchitis plus a few rales and fine crepitations, indicating the present situation of the semi-coagulated blood; in this group our main anxiety is the determination whether phthisis is at the bottom of the matter or not, and the question will be found discussed under that heading (*vide* pp. 54 and 92).

We will now assume that (2) the blood has come from some part of the alimentary system. We have to consider the—

Causes of Hæmatemesis

	Diagnostic Points.
Swallowed blood (rarely of noticeable extent).	Already considered; local examination usually sufficient to clear up the case, except in a few cases of malingering, with deliberate drinking of blood.
Vicarious menstruation (very rare).	Of very doubtful occurrence; can only be thought of in a girl, probably young and hysterical; no other local symptoms, periodicity of bleeding.
Bloodstates in— Purpura, Scurvy, Yellow fever, Acute yellow atrophy of liver, etc.,	All rare as causes of hæmatemesis. — The condition of the patient induced by the named disease usually quite sufficient for diagnosis.
Traumatism.	History sufficiently obvious, either of an external blow or of swallowing a corrosive or violent irritant, or some mechanical lacerator of mucous membrane.
Gastritis or simple vomiting.	Hæmorrhage slight as a rule, and usually only in streaks; the history of severe vomiting preceding the blood usually sufficient, except in those cases where the violence has actually ruptured a large vessel; *vide* below, Aneurysm and Ulcer, etc.
Aneurysm.	Quite possibly unrecognised till post-mortem is made; only diagnosable during life when

	Diagnostic Poi
	previous examination has revealed a tumour of abdomen with expansile pulsation. Hæmorrhage very profuse, and probably rapidly fatal.
Cancer.	Hæmorrhage usually of coffee ground type, great loss of flesh, and probably a tumour ; patient usually beyond early middle age.
Ulcer.	Hæmorrhage usually free, with clots ; in simple ulcer, patient usually a young person and more commonly a female. I have known a tubercular ulcer in a phthisical patient cause fatal hæmorrhage, but such cases are very rare.
Diseases of other organs leading to venous congestion—	
(*a*) Morbus cordis.	Presence of bruits and other evidence of cardiac back pressure.
(*b*) Chronic pulmonary disease.	Evident physical signs of lung disease, associated with failure of circulation.
(*c*) Cirrhosis of liver.	History of alcohol, or dyspepsia and vomiting suggestive of alcohol ; liver enlarged probably.
(*d*) Portal congestion.	Produced by ascites, carcinoma of peritoneum, enlargement or diseases of spleen, etc., with tolerably obvious features.

Such is a practically complete list of the causes of the presence of blood in the stomach leading to hæmatemesis. In numbers they look formidable enough on paper, but in practice they are comparatively simple in diagnosis. Thus, hæmorrhage from the stomach is *never the first and totally unexpected* symptom of serious bloodstates, nor of those forms of back venous pressure commonly attributed to portal congestion. There are and have been some prodromal or leading indications in all cases, the hæmatemesis forming a culminating calamity, so to speak, not perhaps expected, but not altogether unforeseen. There is only one group of cases that really presents much difficulty, viz. those in the middle period of life, especially in men, where we have to try and decide between cirrhosis of the liver, carcinomatous or simple ulceration, and an unexplained cause which provisionally may be termed simple varicosity of veins. More than one such case has occurred to me in which age and sex, history of alcohol, slight dyspeptic phenomena, and the entire absence of positive objective signs (enlarged liver, tumour, dilated stomach, etc.), have together formed a

symptom-complex, in which the diagnostic points have been so nicely balanced that a positive diagnosis has been impossible in the absence of a post-mortem. Luckily for the patient the diagnosis, from a therapeutical point of view, is here of comparatively little moment, for absolute rest in bed, combined with physiological rest for the stomach, constitute the main, if not the only means of combating all form of serious gastric hæmorrhage. For some further indications, *vide* under the headings of Ulcer, Cirrhosis of Liver, etc.

Anal Appearance of Blood

We must now consider blood passing from the bowel by the anus. It may be laid down as a working rule that blood in a comparatively unaltered state, *i.e.* recognisable as such by the laity, passed per anum comes from the colon or rectum, or the lowest two feet or so of the ileum. Blood coming from the stomach or upper part of the small intestine will be so altered by the digestive processes as to appear as melæna or black tarry stools. The only exception (it occurred to me once in tubercular ulcer of the stomach, verified post-mortem) is a case in which the bleeding is so profuse as to practically fill the intestine, and so appear at the anus simply darkened in colour. Such cases are almost invariably fatal.

The following are the Causes of Melæna

Blood passing from the stomach.	These cases constitute an enormous majority (probably 90 per cent) of cases of melæna; hæmatemesis is very likely to have occurred; for differential diagnosis, *vide* table above.
Duodenal ulcer, or simple, tubercular, or malignant disease of small intestines.	Rare as causes of melæna; suggested chiefly by the presence of the symptom (melæna) combined with the absence of the commoner causes; possibly some tumour felt or some abdominal symptoms suggesting ulceration, but not of a gastric type.
Anchylostomiasis.	Very rare indeed in England as a cause of melæna and anæmia; only to be detected by finding eggs in the fæces.

Causes of Hæmorrhage (recognisable as such by the Laity) passing per Anum

	Diagnostic Points.
A. Lower ileum— Ulcers: Typhoid.	Hæmorrhage appearing in course of a definite attack or of a vague pyrexial illness, when the hæmorrhage clears up the diagnosis.

	Diagnostic Points.
Tubercular.	Usually distinct indications of tubercle, but diagnosis often impossible, *ante* mortem, from typhoid ; *vide* Typhoid *v.* Tuberculosis.
Dysenteric.	History of recent or old dysentery fairly obvious.
Simple and carcinomatous.	To me unknown causes of severe hæmorrhage, but might conceivably give rise to the symptom, but would not be diagnosable, except by absence of above three troubles, and in carcinoma possibly a tumour.
Traumatism.	History of a blow or swallowing a foreign body. If such a case arose it would be quite a pathological curiosity.
Drugs, especially violent purges.	History of administration.
Blood conditions.	The same as in the stomach, but all rarer ; occasionally has occurred as a sequel to the very debilitating scourge of influenza ; such was the cause of death of a lately deceased distinguished member of our profession.
Intussusception.	Signs of intestinal obstruction, presence of straining, etc.
B. Cæcum, colon, and sigmoid flexure.	All similar causes to those of ileum, but dysenteric and carcinomatous ulceration are here very common causes of hæmorrhage instead of being rare, while the reverse is the case with typhoid and tubercular troubles, intussusception commencing in colon very rare indeed.
C. Rectum— Traumatism. Piles. Polypi. Prolapse. Intussusception coming down. Ulcers—dysenteric, malignant, etc. Strictures. Fissures, fistulæ, etc. Vaginal sources, to be looked for in females.	All within reach of the examining finger or speculum, so that their diagnosis requires no discussion ; examination of the rectum also discloses the fact that the blood does come from the lowest six inches of the bowel.

Like hæmorrhage appearing by the mouth, the causes of melæna and rectal hæmorrhage are much more formidable on

paper than in practice, and it is but very rarely that some clinical features will not be found leading unmistakably to a definite diagnosis.

VOMITING

Vomiting is a very complex process, of which the actual emptying of the stomach forms only a culminating objective feature. According to physiology, the whole series of phenomena ending in emesis is best explained by the hypothesis that there is situated in the medulla oblongata a group of neuron cells (termed a vomiting centre), the dendrites of which are connected with the nervous system in such a way that, on the one hand, they may receive afferent impulses from any part of the body, which impulses are capable of stimulating them to special activity, and, on the other, they may transmit messages to the salivary glands, to the respiratory mechanism, and to the voluntary muscles which, when thus stimulated, produce the emptying of the gastric contents. By accepting this explanatory hypothesis, we are in a position to gain a more or less rational understanding of the innumerable pathological or clinical incidents that may be associated with vomiting. The afferent impulses (as from a crush or severe pain, however excited) from any part may be so strong as to cause it, or, on the other hand, the condition of the blood circulating through the centre, or the physical condition of the brain, may be such as to independently start the centre into action. Lastly, and far most frequently, the contents or condition of the stomach itself may start the afferent impulses.

A purely scientific or physiological analysis of vomiting should therefore start with a division into central or direct, and peripheral or reflex, each with its subdivisions; but from a clinical and diagnostic point of view a more useful classification is into the following:—

Causes of Vomiting

Diagnostic Indications

Affections of nervous system. — Cerebral—Tumour. Meningitis. Cerebritis, etc. General shock.	Not usually associated with much nausea, independent of food; almost invariably associated with marked corroborative evidence—optic neuritis, fits, paralysis, etc.—may be quite intermittent for even long periods, and then its apparent causelessness is its great feature.
Affections of nervous system. — Gastric crises of tabes dorsalis, or G.P.I.	Apparently quite causeless in its onset, and equally mysterious in its cessation after a day or two's duration: also independent of food, associated with more local pain than cerebral form.
Affections of nervous system. — Hysterical.	Probably in a young person, and one who is not losing flesh, notwithstanding the assertion that for months everything has been vomited.
Affections of nervous system. — Cardiac disease.	Distinct evidence of bruits and back pressure on other organs than the stomach.
Pulmonary diseases attended by cough — phthisis, etc.	On inquiry patient will say, "I cough till I am sick." Physical examination rarely fails to show good cause for the cough, but it is astonishing how patients will complain of the vomiting and forget the cough till a leading question on it is put: "Are you sick because you cough, or are you sick independently of coughing?"
Acute infective diseases—the zymotics, pneumonia, etc.	Although vomiting, especially in children, is common at the onset of febrile acute illnesses, it must be very rarely that the cause is not soon obvious, for the thermometer will rapidly remove doubt.
Uræmia and other kidney troubles.	Vomiting again causeless, and independent of food; urinary examination soon clears up the case, even if it be unattended with commoner symptoms of uræmia.
Simple pain.	Statement made by patient quite sufficient.

Diseases or conditions in abdomen.	Pregnancy, tumour, obstruction, etc.	Physical examination of the abdomen very soon clears the matter up; abdominal pain or discomfort usually marked.
	Addison's disease.	Vomiting usually associated with pain, but the accompanying physical weakness, and especially the almost imperceptible pulse, will point to the disease even if typical bronzing be not present.
	Gastric trouble— Ulcers. Irritation. Inflammation. Pyloric obstruction, etc. etc. Poisoning.	Vomiting due to actual disease of the stomach is, broadly speaking, characterised by being in some definite relationship to food and to pain or discomfort produced by food. For other points separating the various gastric troubles, *vide* Gastric Ulcer, etc.

If we are called upon to investigate a case of vomiting, very little observation is required to eliminate certain of the above groups—the pyrexia and aspect of acute general diseases, the collapsed features or the history of obstruction, the evident dropsy of morbus cordis, the pain or shock of an accident, etc., will at once put even the most careless observer on the right track. The diagnostic difficulties begin when the vomiting is the sole obvious feature that attracts the anxious attention of the patient or of the friends in the case of children.

In quite young babies, *i.e.* in the first year of life, it is almost safe to conclude at once that food and consequently gastric irritation, is at the bottom of the mischief, though we must not forget to look for signs of congenital syphilis. After the first year up to, say, puberty the insidious onset of tubercular meningitis with causeless vomiting must never be absent from our minds, however obvious may seem at first sight the apparent cause for the symptom. It is also during this period of childhood that all acute illnesses are apt to have vomiting for a prominent feature in their early stages, from which fact we may safely deduce as a golden rule — *No case of vomiting in a child can be safely despised till a few days have passed without the development of any more guiding symptoms.* It may, however, be said that in acute gastric conditions food will be at once rejected, while in all other forms of sickness the food will usually stay down for some little time, and that without pain or discomfort.

In adults we may draw attention specially to "morning vomiting"; this, when repeated, is almost confined to the following conditions: pregnancy, alcohol and alcoholic dyspepsia; occasionally the vomiting

of uræmia and of cerebral tumour assumes this type, as almost invariably does the vomiting which is brought on by the cough of chronic bronchitis. Once reminded of this peculiarity diagnosis is not difficult.

COLIC

Strictly speaking, the terms "colic" and "colicky pain" should be reserved for intermittent or at least remittent painful sensations in the abdomen, or referred to the abdominal viscera; but I wish here, for convenience' sake, to use the expression in a more comprehensive sense to include all forms of severe pain referred by the patient (either by voice, or gesture in those unable to speak) to the abdominal or pelvic region. Pain thus referred may arise from disease of any structures of the trunk below the thorax, for none of the viscera were intended for organs of special localising sense, so that pain arising in one part is frequently misreferred to another spot. For this very reason the differential diagnosis of the various causes for the symptom assumes here a greater importance than in any other region, especially as the treatment for one cause of the pain would be absolutely and rapidly fatal if another cause were at work, *e.g.* perforation of stomach *v.* a gall-stone.

The causes, then, are many and various. They may be divided most simply into groups:—

CAUSES OF ABDOMINAL PAIN

Group I.—*Affections of the Peritoneum and Alimentary Canal itself*

(*a*) Irritating contents of the gut causing either congestion of mucous membrane or painful peristalsis, or more likely both together.
Food unsuitable in quantity or quality.
Foreign bodies of any description.
Drugs, active purgatives, or poisons.

(*b*) Ulceration and other disease of the walls; simple ulcer or tubercle, or carcinoma, etc. (*N.B.*—It is astounding how frequently such diseases exist without causing any *pain directly* by their mere presence.)

(*c*) Embolus of a mesenteric artery I have known to cause intense agonising colic with other symptoms like obstruction.

(*d*) Obstruction and strangulation of the gut, whether acute or chronic, and however arising (intussusception bands, hernia, twists, etc.).

(*e*) Peritonitis, acute or chronic, local or general, however arising, independently or through—

(*f*) Perforation of any hollow viscus, or rupture of a solid one.

(*g*) Some blood alterations or blood-carried poisons must be inserted here; of these lead and gout are the only two of practical importance.

(*h*) Nervous disturbance of gut from independent disease or functional condition of cord or brain; *e.g.* the gastric crisis of tabes, or of G.P.I.; or disease of the suprarenals; or abdominal nerve complexus as met with in Addison's disease, etc.

Group II.—*Affections of Organs in Direct Connection with the Gut*

(*a*) Liver:—
Inflammation or abscess, primary or arising in connection with a previous painless affection.
Carcinoma and other rapidly increasing growths.
Gall-stones when trying to leave the gall-bladder.
Rupture (only traumatic).

(*b*) Pancreas:—
Acute inflammation.
Stone in the duct (very rare, and practically outside the range of certain diagnosis).

(*c*) Spleen:—
Abscess.
Enlargement, especially if also mobile enough for difficulties to occur in the circulation to and from it.
Embolus (pain rarely very severe).

Group III.—*Affections independent of the Alimentary Tract and its Connections*

(*a*) Of the urinary tract:—
Stone or other painful kidney trouble.
Kinking or disease of ureter.
Bladder: distension or rupture.

(*b*) Abscess from any source; its presence or its rupture may cause acute pain.

(*c*) Aneurysm: its presence or rupture.
(*d*) Affections of the bones of the vertebral column, especially caries.

Group IV.—In women the pelvic reproductive organ must be borne in mind as the possible primary source of growths, abscesses, peritonitis, bladder disturbance, etc.

In dealing practically with such a heterogeneous collection of causes for acute abdominal pain, it is obvious that we must have some guiding principle on which to work; though a general, and often even a precise and exact, diagnosis is by no means so difficult to arrive at as might at first sight appear. The simplest method appears to me to take events in the chronological order in which they occur, with possible or probable narrowing of the field with each. On a message being received to see a patient in this condition, the first information naturally given is, roughly, the age of the patient, baby, child, or adult. We may say at once that in subjects under puberty direct alimentary disturbances are almost alone to be thought of—intestinal obstruction or strangulation, simple colic, possibly appendicular trouble or peritonitis, or may be even a renal stone—but such cases as tabetic crisis, Addison's disease, aneurysm, gall-stone, etc., may be at once excluded. After puberty no such general rule can be laid down, though it is true that certain troubles, *e.g.* gall-stone and malignant obstruction, are much more frequent in stout elderly patients than are others, *e.g.* intussusception or perforating acute ulcer.

The next step, as things occur in practice, is to inquire of the friends as to the previous illnesses of, or similar attacks experienced by, the patient, and also as to symptoms additional to the pain:—

Is this the first attack?

A. If not, has the patient previously—

Been jaundiced?
Passed gravel?
Had urinary trouble or difficulty?
Complained of anything suggestive of G.P.I. or tabes?
Suffered from marked irregularity of bowels?
Lost much flesh, or rapidly emaciated?

B. If it is, How did it come on? Was it—

(*a*) More or less gradually in the course of a fairly definite

illness, or at least has the patient been complaining long?

e.g. Typhoid. Gall-stones.
Addison's disease. Gastric ulcer. Dyspepsia.
Phthisis.
Gout.

or, (*b*) Suddenly, in the midst of perfect health?

e.g. Latent gastric ulcer with perforation.
Hernia or other strangulation of gut.
Appendicular trouble.
Indigestible meat, poisons, etc.

The answers to these questions, and independent statements by friends, will very probably have thrown great light upon the case, excluding certain causes almost to a certainty, and bringing others forward into equal prominence as probabilities. Any symptoms additional to the pain will also very likely throw great light in certain diagnostic directions. Those more particularly to be asked for if information is not volunteered are—

Vomiting.
Constipation or other state of bowels, such as straining at stool, or diarrhœa.
Strangury, or frequency of micturition, or other obvious urinary trouble.
History of blow, or other accident.

After gaining as much information as possible from what may be called external sources, the next step is to visit the patient and make as careful an examination, verbal and physical, as circumstances will permit.

We will now for a moment leave diagnosis itself, to consider the object we have to gain by it, viz. correct treatment of the condition.

Treatment of acute abdominal pain must proceed on one of four lines:—

A. Absolutely expectant, except for the outward application of hot fomentations, or an ice bag;
B. A hypodermic injection of morphia, or inhalation of general anæsthetising agents, such as chloroform or ether;
C. An emetic or purge;
D. Prompt surgical interference: manipulative or by the knife;

and it is primarily the object of diagnosis to determine upon which of these lines we may most safely proceed.

The last is out of all proportion the most important from every point of view, so we will first investigate the symptoms which indicate that a condition is present which demands this line of treatment. The conditions themselves are :—

1. Perforation or rupture of any hollow or solid viscus (gut, bladder, uterus, liver, etc.), or of any pathological tumour in the widest sense of the word (aneurysm, cyst, abscess, etc.).
2. External strangulation (strangulated herniæ of all sorts palpable externally), or internal strangulation of a viscus or tumour by twist of its pedicel, or by band, etc. (internal herniæ, etc.).
3. Acute general peritonitis of unknown origin (as well as most cases of known definite causation).
4. Acute pancreatitis and embolus of a mesenteric artery, which cause symptoms precisely resembling those of strangulation of the gut, and can only be distinguished on operation.
5. Distended bladder.
6. Displacements of pregnant or pathological uterus, causing urgent symptoms.

Luckily most of these conditions are, even in their earliest stages, usually associated with such diagnostic features as to be at once recognisable as a group of cases. Thus the—

Pain.—Is very sudden in onset, rapidly becoming agonising in character, may be continuous or intermittent, and in many external hernial cases so correctly localised as to direct attention at once to the seat of trouble.

Aspect of the Patient.—Is frequently distinctive, with ashen pallid face, and eyes deeply sunk, and pinched features; collapse very profound, with cold and clammy sweat.

Pulse.—Is small, thready, and frequent.

Tongue.—Is very dirty, and with great tendency to dryness.

Vomiting. — Is often incessant, most distressing, very rarely indeed quite absent; if any delay has occurred may be stercoraceous.

Constipation. — Absolute since the onset of pain; thus distinguishing such cases from virulent poisons, in which diarrhœa is present with vomiting.

Abdomen.—Is very tender to touch as well as painful, and may be already distended or rapidly becoming so; evident laboured waves of peristalsis may be present and give useful information.

Urine.—May be much diminished, or totally suppressed without strangury.

Should all these indications be present, or even two or three of them in well marked form (especially small, thready pulse, dry tongue, and incessant vomiting), there can be no room for hesitation if life is to be saved—prompt surgical measures must be adopted, manipulative or by cutting, according to circumstances. It however only too frequently happens that the symptoms are not so well marked at the onset, and diagnosis must perforce be postponed for an hour or two. The only safe and golden rule is then: "In any case presenting the above features in doubtful form, but where suspicion is in the slightest degree aroused, no morphia must be administered"; for thereby many of the most important developments are masked or prevented; but the patient must be placed under intelligent observation with hot or cold abdominal applications, and his case must be reinvestigated in an hour or two, by which time the diagnosis will have become more certain. Measures may then be taken accordingly.

A distended bladder, though possibly a cause of acute abdominal pain, ought not to be difficult of diagnosis; if the distension is sufficient to cause pain, the viscus will always be easy to feel as a centrally placed abdominal tumour. A bladder ruptured through disease, though probably causing symptoms belonging to (1) (where, indeed, it has been placed), will have invariably been preceded by a long history of urinary trouble and difficulty, and will have thus been diagnosed.

The pregnant uterus will likewise not offer, under these circumstances, any diagnostic difficulty. Cases of rupture come under (1), and the blanched, bloodless aspect of the collapsed patient will be suggestive in a woman known or suspected to be pregnant.

Into the more definite diagnosis of these cases I do not propose here to enter: the keenest diagnostician must frequently content himself with saying, "Laparotomy is urgently indicated," leaving the operation to disclose the exact condition. Thus, acute pancrea-

titis presents all the features of acute intestinal *strangulation*, and acute perforative peritonitis is often identical in its symptoms with volvulus or internal hernia.

Abdominal Cases without Marked Collapse

Collapse developing very early in the case (almost from the instant of onset), with its attendant facies, pulse, etc., we have hitherto taken as the guiding principle in diagnosis. We have now to consider those cases of abdominal pain in which it is not so marked, may be even absent, at any rate in the earlier stages. Such cases, especially if on examination no pathognomonic sign is present, still call for the keenest eye, clearest understanding, and most incessant watchfulness, that at the earliest moment the cloven hoof betraying serious developments may be detected—something more than pain written in the face, some smallness and frequency of pulse out of proportion to the expected; a persistency of vomiting, or alteration in the character of the vomited matters—any or all of these features may be found on a second visit.

In no case of painful abdominal affection can it be said that collapse must be absent, but it is, at any rate in severe shape, an exceptional feature in otherwise fairly healthy people (at the extremes of life or in broken down subjects great caution in judgment is necessary), when one of the following is present, causing complaint of abdominal pain:—

Simple colic, from improper contents of gut, or due to lead, gout, or Addison's disease, or nervous troubles;
Liver diseases, apart from a possible suppurative peritonitis arising from it;
Chronic obstruction of bowels;
Typhlitis;
Aneurysm;
Caries of spine;
Retroperitoneal growth;

and we shall now proceed to discuss their differential diagnosis on the assumption that the patient is capable of giving intelligent answers to questions, and that a thorough examination of the abdomen is allowable and allowed.

On approaching the patient definite jaundice, if present, will at once attract notice (*N.B.*—by artificial light this is very easily over-

looked, in fact, impossible to detect, and the question of its presence must always be asked), and direct attention to the liver as in some way the cause of the trouble. The discovery of jaundice would also lead to questions as to its previous occurrence, if such had not already been put. The pigmentation of Addison's disease would at this stage possibly attract attention, and if present go far towards the elucidation of the diagnostic problem.

General inquiries to establish points left doubtful by the friends may then be put to the patient; their scope has been already indicated. The abdomen must then be exposed, and the patient asked to point out the seat of greatest pain, and the direction (if any) in which it seems to travel; this point is often useful, but as already noted, too much stress must not be laid upon it, owing to the vagaries of referred pain.

The orderly methodical examination of the abdomen by inspection, palpation, and percussion, and even by the stethoscope, may be proceeded with, and if there is still a doubt, a vaginal or rectal examination, or both if necessary, must be made.

This examination will almost for a certainty have revealed any of the following points which are present :—

1. Whether the pain is greatly increased by pressure, or somewhat relieved as a steady pressure becomes firmer and deeper; the former suggestive of peritonitis or serious organic disease, the latter of simple colic.

2. Hardness or increased resistance of the muscles, either locally or generally, suggestive of some inflammatory trouble beneath them.

3. Tumour or swelling of liver, spleen, kidney, or bladder, or independently existing in abdomen, suggestive of its being the real seat and possible cause of the pain, or indirectly affording an opportunity for obstruction or strangulation of the gut.

4. *Per vaginam*, fixity, or swelling of uterus, or other palpable abnormal condition of female reproductive organs again directly or indirectly causing the mischief.

5. *Per rectum*, ballooning, or the peculiar smell of carcinoma (not easy to describe, but like some other smells once experienced never forgotten) or definite stricture, or tumour within reach of finger, all suggestive of carcinoma or other serious rectal disease, or intussusception reaching the rectum.

6. In cases still obscure we shall have tried to elicit pain on flexing thigh against resistance, suggestive of renal or appendicular or psoas mischief, or tenderness on tapping or jarring spine, suggestive of caries or other serious bone mischief.

Founded upon these and similar features, we may epitomise the principal points of differential diagnosis in such cases as follows:—

Differential Diagnosis of Causes of Abdominal Pain not usually associated with Marked Collapse, or urgently demanding active interference

Colic, Simple or Functional, of Intestine

Probable history of an over-full, indigestible, or unsuitable meal; pain probably relieved or not made worse by pressure; vomiting severe, but not prolonged, and diarrhœa probably present, or soon sets in.

Lead and gout must be thought of in adults as a possible cause, and a blue line on gums or tophi looked for, and inquiries made of occupation, etc.

Tabes dorsalis and G.P.I. must also be remembered in this connection, and the knee jerks and pupils investigated.

After all is said and done, the most important problem is here to separate these cases from commencing local or general peritonitis, and from forms of intestinal obstruction which begin insidiously (overlooking a gall-stone or renal calculus is less serious, but *vide* below), and the vital importance of the subject must be my excuse for repeating and bringing into closer contrast the likenesses and differences of the two conditions.

Besides the mere pain itself, then, they may be alike in—

1. The vague localisation of the pain.
2. The suddenness of its onset.
3. The universal tenderness of the abdomen on light palpation.
4. The intensity of the pain.

But they more frequently and essentially differ in—

	Functional colic.	Obstruction and Peritonitis.
Locality.	Very vague, usually epigastric or umbilical.	Not unfrequently correctly localised.
Suddenness.	Gradually rising to a maximum, and waning and waxing.	Most commonly absolutely sudden, and then persistent at a high pitch.
Superficial tenderness.	Often great, in fact, greater than when	May be great, but is never greater than

	Functional Colic.	Obstruction and Peritonitis.
	fairly firm pressure is applied, which may completely check the pain.	that caused by firm pressure.
Collapse, including sunken eyes, small pulse, dry tongue, clammy sweat, etc.	Rarely present, except at extremes of age and in broken-down subjects.	Usually comes on at once, and in any case develops very rapidly; only in the rarest of instances is it quite absent.
Vomiting.	Rare, except in acute poisoning by drugs or food, and then never stercoraceous, and only excited by food.	Very common indeed, usually very persistent, and may be stercoraceous, independent of food.
Constipation.	Not common, except in lead poisoning.	Usually complete (even to wind) after the onset of pain; bowels may have been open just previously or pain may come on during defæcation.
Diarrhœa.	Far more frequent than constipation, except in lead colic.	Only exists in the shape of tenesmus, with passing of mucus and blood. *N.B.—In suspicious cases, when diarrhœa is complained of, examine the alleged motions.*
Abdominal swelling or tympanites.	Practically do not occur, at any rate early in the case.	General distension frequent, and forms a very suspicious feature indeed. May arise very early in the case.
Muscular rigidity.	Easily overcome, if present, by gentle, steady, persistent pressure.	May be quite local, but is usually present, and cannot be overcome, any effort to do so causing great increase in the pain.

In the remaining conditions causing severe abdominal pain,

immediate diagnosis on the first or even second visit is of less vital consequence, though important enough, as it must essentially be, if medicine is to maintain any grounds of claim to rank as a science.

Affections of the Liver

Simple non-purulent inflammation or congestion.	Two cases have recently come under my care in which I believe this condition was present; the pain was localised to the right hypochondriac and epigastric region, and both were associated with a distinctly appreciable liver edge, which was very tender on pressure or manipulation; in both alcohol was much in evidence, and both subsided in a few days without further symptom.
Suppuration.	Probable history of dysentery or tropical residence; pain localised correctly in liver; frequently a swelling to be made out; a hectic type of temperature, and especially if *associated with night sweating*, is very suspicious.
Carcinoma.	Easily diagnosed if tumour is felt; if no tumour, then rapidity of loss of flesh is most important feature, when the locality of pain, and age of patient, rouse a suspicion.
Gall-stones.	Possibly previous history of jaundice; patient usually stoutish and middle-aged or older; distended gall-bladder, if present, almost conclusive.

It is usually assumed that gall-stones, renal calculus, and simple colic are very likely to be mistaken for one another. The following table shows their contrasts and likenesses:—

Gall-stones, renal calculus, simple colic may be alike in—

1. Sudden onset of pain.
2. Sudden cessation of pain.
3. Indefinite locality of it.
4. Severity of it.

They more commonly differ in–

	Gall-stones.	Renal Calculus.	Simple Colic.
5. Locality and—	Usually referred more correctly than the others to the right hypochondrium.	Usually in back, loin, and groin.	Usually referred to umbilicus or epigastrium.
6. Direction of pain.	Pain fixed, or seems to pass upwards.	Travels down groin to testicle, which is frequently retracted.	Fixed in umbilicus, or travels across abdomen.
7. Character.	Not intermittent.	More intermittent than gall-stone.	Usually is actually intermittent.
8. Calculus found.	In fæces if at all.	In urine if at all.	None.
Associated symptoms, if any.	Jaundice; urine dark coloured perhaps; otherwise the function not interfered with; vomiting may occur from the pain or associated gastric disturbance.	Strangury or frequent micturition; possibly bloody urine; function pretty certainly interfered with; vomiting less likely than with biliary colic.	Urination not interfered with, except possibly quantity diminished if much vomiting, which is more probable than in the other two.
Previous history.	Of jaundice and similar attacks.	Of gravel in urine, or other pathological condition of urine.	Of "bilious attacks," if anything, or of dropped wrist.
Age.	Middle or later.	Any age.	Middle and young most likely.

Chronic Obstruction of Bowels.—Previous history of notable irregularity of bowels; rectal examination may give very important information; possibly a tumour in known line of colon (malignant or intussusception).

Typhlitis.—Pain usually correctly referred to right iliac fossa; dulness on percussion and great tenderness, especially at

M'Burney's point. If definite swelling and redness diagnosis nearly conclusive.

Aneurysm.—Tumour has expansile pulsation; pain more in back and worse at night; bruit possibly heard over tumour. Vessels elsewhere degenerate: history of syphilis and hard work and alcohol probably.

Caries.—Pain also referred to back, probably worse on jarring heels or tapping spine; this or aneurysm may cause definite cord symptoms.

Retroperitoneal Growth.—Only guessed at by excluding other causes for pain or cord symptoms. Possibly a tumour of irregular outline may be felt, lying deeply, and without expansile pulsation.

DIARRHŒA AND CONSTIPATION

The causes of these conditions or symptoms may thus be tabulated:—

	Clinical Illustrations.
1. Contents of the gut.	
Too liquid.	Doubtful if this condition *per se* ever causes diarrhœa, possibly excessive drinking might do it; unduly active peristalsis may thus cause it by preventing absorption.
Too solid.	Probably unusual sweating, habitual neglect of the bowels; fever thus possibly causes constipation by too great absorption of water.
Too irritating.	Improper fermentation, unripe fruit or other indigestible material.
Foreign bodies.	Either by their presence cause irritation possibly, or by their size cause obstruction, *e.g.* purgative drugs, or gall-stones, etc.
2. Walls of the gut.	
(*a*) Ulcers: *if not too deep to paralyse.*	Tubercular, typhoid, dysenteric, and simple.
(*b*) Growths.	May cause irritation, and so diarrhœa, or paralysis, and so cause constipation; sometimes by bulk or cicatrisation may also cause obstruction.
(*c*) Inflammation.	of peritonitis leads to paralysis, the most

	Clinical Illustrations.
	obstinate form of obstruction; milder forms of this may by irritation cause diarrhœa, as in some cases of appendicular or cæcal trouble.
(*d*) Altered structure.	Lardaceous disease, leading to obstinate diarrhœa.
(*e*) Altered circulation.	Heart disease, cirrhosis of liver, either constipation or diarrhœa.
(*f*) Blood conditions or toxæmias, which may either cause excessive peristalsis or excretion into gut, or both, or, *per contra*, may act in the reverse manner.	Bright's disease (uræmia); gouty habit; probably some of the diarrhœas of zymotic cases, critical or otherwise, are thus caused. Lead is the best known example of the opposite condition, viz. constipation.
(*g*) Reflex nerve influence, again acting either on peristalsis or secretion, or both.	Cord diseases; mental states, as fear, anxiety, insanity, etc., dentition in babies, commonly causing diarrhœa, but not unfrequently the reverse; and here possibly may be placed the too solid motions of habitual neglect.
(*h*) Pressure from without acting mechanically or reflexly.	If an abdominal tumour presses on the gut it is likely to cause constipation, but sometimes it may cause diarrhœa reflexly, or by direct irritation.
(*i*) Some unexplained causes.	*e.g.* To what shall we attribute the almost universal constipation of the early days of sea-air, or other changes of climate and temperature?

The majority of the above cases offer no difficulty in diagnosis, for the constipation or diarrhœa is merely a concomitant symptom of some obvious disease or departure from the ordinary habits—dietetic, physical, or general—of the individual; but a few guiding rules of procedure are required for the elucidation of those cases in which the diarrhœa or constipation is the only obvious symptom from which the patient is suffering, or, at least, of which he complains.

In any such case the first and most important thing to ascertain is the precise meaning attached by the patient to the words he uses—ask how often the bowels act, whether much tenesmus or not, and

the quantity and character of what passes, and inspect the motions for this purpose if necessary. A case recently has occurred to me in which the patient complained of an obstinate diarrhœa. Somewhat puzzled by my ineffectual efforts at drug treatment, I was led to ask him to save everything passed for twenty-four hours; I then found all the motions solid and natural, and further inquiry led to the conclusion that it was simply a case of a gross feeder whose bowels required naturally a more frequent relief. This inquiry and inspection will probably result in the discovery of any of the following conditions of the stools:—

Natural in Consistency and Quantity.—As in the above case. If motions very narrow and ribbon-like, probably a stricture, organic or spasmodic (of sphincters), is present.

Much Feculent Material, but too Watery, Light, or Dark.—If light or natural colour, suggestive of simple irritation of upper bowel by unsuitable food; if dark and tarry, strongly suggestive of hæmorrhage (*q.v.*), high up.

Masses of Undigested Food or Curds (in Children), so-called Lientery.—Suggestive primarily of unsuitable or too free feeding, *e.g.* too much milk in typhoid; and if this question is satisfactorily determined in the negative, then suggestive of undue nervous influences, perhaps best treated by bromides or nervine tonics.

Pea-soupy Stools.—Might rouse a suspicion of insidious typhoid, and should lead to the use of the thermometer.

Scybalous Knobs or very hard Fæces; probably with much Mucus.—Suggest a *false diarrhœa*, due to undue constipation; hardening of the fæces; a most important class of cases, because they are so common.

Pus, or Pus and Blood; Mucus, or Blood and Mucus.—Rupture of an abscess into bowel. Much mucus is a pathognomonic sign of colonic trouble, and if present with hardened lumps almost certainly indicates constipation, either simple or due to organic obstruction, as the real trouble; if with blood it is suggestive of dysentery, malignant or other stricture, piles, fistula in ano, etc.

Membranous Masses floating in Water.—Membranous colitis; in the absence of an acute illness rather suggestive of malignant disease, or occasionally chronic obstruction or simple constipation.

The anus at least, and better also the rectum, should then be

inspected and examined to see if any pathological condition is there present which can cause diarrhœa, or at least explain the patient's complaint, thus :—

Piles or Simple Polypi.—May explain blood and mucus, excessive straining pain, etc.

Fistula.—By its discharge may have given a false impression of diarrhœa, as well as altering the material of a stool.

Ballooning.—If well marked is strongly suggestive of a stricture just out of reach, or that can with an effort be just touched.

Stricture.—Malignant, syphilitic, etc., will explain either diarrhœa or constipation, or a marked alternation of the two conditions.

Rectum full of Hardened Fæces.—A very common thing to find in complaints of either diarrhœa or constipation. It must be emptied, and then a fresh examination made, as the condition may be a simple one, or it may be merely a symptom of some serious trouble to be discovered by the examination of the empty viscus.

Uterine and Ovarian Troubles.—In women a rectal examination is often required as well as vaginal; it is possible for almost any organic disease or pathological position of the genitory organs to cause a disturbance of defæcation.

A systematic examination of the abdomen, which may precede or follow the rectal one, may very likely reveal an efficient explanation of the trouble. Almost every pathological condition of the abdominal viscera may cause alteration in alvine evacuation, *e.g.* liver diseases (*q.v.*), splenic enlargements, any abdominal tumour, dilated stomach (*q.v.*), tubercular peritonitis, or any other form of peritonitis, or ascites, etc.; as, however, most if not all of them will have given rise to complaints other than of diarrhœa or constipation, they will not here be further considered.

Should the history and examination thus far have left matters still in doubt, the patient must next be thoroughly overhauled for evidence of some general disease, or a local one not situated within the abdominal region, which may be associated as cause with the symptoms under consideration. These are chiefly phthisis, lardaceous disease, plumbism, Bright's disease, typhoid, leucocythæmia, and other blood dyscrasias or definite disease of the spinal cord or brain.

These, like the troubles mentioned in the preceding paragraph, hardly require in this connection any further mention. The chronic

cachexia, or suppuration, with the enlarged spleen and urinary condition of lardaceous disease; the blue line, colic, and occupation of plumbism; the temperature of typhoid; the blood of leucocythæmia and anæmia; the low specific gravity *and* diminished urine of approaching uræmia (the diarrhœa is really a uræmic phenomenon); these are all sufficiently distinctive; and so are the physical signs in the chest of a phthisical patient; but it must not be forgotten that in consumption there are several possible sources for the diarrhœa which require a little consideration, owing to their importance; thus:—

Causes of Diarrhœa in Phthisis

(*a*) Accidental, as it were, due to food, or change in climate, etc., from which phthisis confers no immunity. This may be sharp while it lasts, but is quite temporary, rapidly ceasing or yielding to treatment. Some change in residence or diet will probably give the clue to its origin.

(*b*) In association with the dyspepsia, which is such a troublesome accompaniment of phthisis, particularly in its earlier stages. In fact, when lung mischief is doubtful but suspected, this dyspepsia is a very suspicious piece of diagnostic evidence.

(*c*) Due to tubercular ulceration of intestine. Usually occurs in later stages, when the lung trouble is very distinct. Associated with slight colicky pains, and very intractable to treatment, though often ceasing spontaneously. The presence of the ulceration cannot be diagnosed with certainty, but pain, and especially tenderness in the abdomen, are suggestive when in combination with an obstinate diarrhœa.

(*d*) Lardaceous changes supervening; painless in itself, absolutely rebellious to treatment; associated with distinctive signs of lardaceous trouble already mentioned in spleen and liver, and possibly kidney.

(*e*) That of exhaustion in the very last stage of the disease; the active cause, in fact, of the fatal ending in many cases not apparently due to either (*c*) or (*d*).

Ascites

The methods to be adopted to determine the presence of excess of fluid in the peritoneal cavity are so distinctly laid down in all the

text-books that I do not propose to discuss them except in tabular form, to separate ascites from cystic formations in the abdomen (*vide* p. 181).

The causes of ascites recognised at the bedside belong to only two categories, or perhaps three, if we include chylous ascites.

A. Irritation of the membrane itself—

By { Tubercle, Carcinoma (secondary), Sarcoma (primary or secondary) } Either of the membrane or of an organ covered by it, *e.g.* ovary, etc.

Inflammatory affections of the peritoneum apart from growths rarely cause marked effusion, unless in conjunction with severe kidney mischief.

B. Undue pressure of blood in the portal system, which may arise from—

1. *Local Causes in Abdomen, practically confined to the Liver and its Fissures, e.g.* cirrhosis of liver, alcoholic or otherwise; perihepatitis; glands or growths (including a gall-stone very rarely) in the transverse fissure, whether primary or secondary.
2. *Produced by a Pathological Condition of other Organs*—

Heart, Lungs, Kidneys { Either secondarily through the right heart, or by extension of growths and inflammation from the pleura through the diaphragm. When due to kidney trouble, it arises probably from alteration in composition of blood, not from pressure.

C. Undue pressure upon the receptaculum chyli or the thoracic duct, leading to transudation of a milky fluid into the peritoneum-chyle.

To determine to which of these primary groups a given case of ascites must be referred is usually a comparatively easy task; to further differentiate the precise individual cause at work is more difficult, and often impossible, without exploration.

In practice one usually first proceeds to exclude Group B 2. The history of the onset of the swelling of abdomen will here give

us very important information—whether, that is, the ascites was the first complaint, or whether the patient had long suffered from other troubles, such as cough, or shortness of breath, puffiness of face or limbs, etc. A careful examination of the thorax and of the urine will then speedily give us either a negative or positive result. If the thoracic organs and the urine are found to present no morbid features, we may be at once sure that some local-abdominal cause is at work. Should any of them, on the other hand, be found in a markedly pathological condition, we know at once that a powerful accessory factor is present in the case, whether local causes are at work or not. The indications from the above examination of the thoracic organs will rarely if ever be of doubtful significance, for ascites does not arise from such causes alone until and unless they are of pronounced severity.

To ascertain the exact local cause at work it is certainly advisable, and, if much fluid is present, often necessary, to empty the abdomen.

In my opinion, this should *always in the first instance* be done by a small surgical incision, and not by a trocar, however small: (*a*) it is more scientific; (*b*) safer for the patient; (*c*) gives more information as to the exact condition of affairs; (*d*) it has been known to cure cases otherwise deemed incurable; (*e*) it allows at one operation removal of a removable cause should such be found.

The character of the fluid (in whatever way it be obtained) will at once detect chylous ascites. A microscopic examination must be made for this purpose, not only to differentiate fat droplets from pus corpuscles, but also for the further intention of deciding the possible presence of cells of heterotopic character. (Physiological injections into animals will, if necessary, detect tubercle.) Should chyle be present, we know at once that we have to deal with a growth, either malignant or glandular, or possibly (very rarely indeed) with a blood parasite—Bilharzia or Filaria.

Should the fluid prove to be clear or purulent, we have to deal with one of the causes in Group A or B (1). Purulent peritonitis is almost invariably associated with (*a*) severe kidney disease, which will have been already detected; (*b*) perforation of a viscus, the occurrence of which will have been only too obvious; or, (*c*) in women, extension from vagina, uterus, or Fallopian tubes, a mode of production always to be suspected in the sex if no other cause is ascertainable. If the fluid has been obtained by incision, this will by itself have probably completed the diagnosis, but if circumstances should have prevented exploration, the following are the principal

diagnostic factors, attention to which, in addition to what has been said already, will usually clear up the case:—

Tubercular Peritonitis.—History of vague attacks of colic and irregularity of bowels. Abdominal walls probably feel thick to pleximeter finger or examining hand; increased resistance. Irregular masses, with equally irregular dulness, may be appreciated. Patient usually young, though (even old) age does not exclude tubercle.

Carcinoma.—Physical signs almost identical with tubercle in the small nodular form, and even on the post-mortem table the two may very closely resemble one another. More commonly the primary growth is obvious as a tumour—ovarian, gastric, renal, etc.

Sarcoma.—If secondary, indistinguishable from carcinoma for clinical purposes. If primary (of omentum) a tumour will be felt giving a sensation to examining hand identical with ballottement of pregnancy. Under thirty-five this cause may be almost at once excluded.

Acute Inflammation.—Recognised by the great pain and tenderness on palpation; board-like muscles (*vide* purulent effusion above).

Cirrhosis of Liver.—Admitted history of alcohol, or if not of alcohol then of morning vomiting; possibly piles or hæmatemesis. Liver usually enlarged, but surface regular, or at least without large bosses of carcinoma.

N.B.—Youthful age does not exclude this cause, unfortunately.

May be a history of syphilis, which points either to cirrhosis, gumma in portal fissure, enlarged glands, or perihepatitis.

Perihepatitis.—To me, as an independent affection this is unknown, but I have seen it in connection with chronic peritonitis of tubercular or malignant origin, and also with syphilis and with heart disease. It is to be thought of when in a case of morbus cordis ascites is *the prominent* back pressure symptom, but I do not believe that it ever arises or causes trouble as a primary and separate disease.

Glands and Growths in Portal Fissure.—If they cannot be felt, will only be diagnosed by a process of exclusion of other troubles; but *when the cause of ascites*, can usually be felt after withdrawal of the fluid.

	Free Ascitic Fluid.	*v.*	Cystic Tumours, or Encysted Fluid of Ordinary Origin.
Inspection of abdomen.	Symmetry usually maintained; flanks usually bulge markedly.		Usually some definite asymmetry to be perceived; flanks do not bulge particularly.
Palpation.	No tumour to be felt, or if so it is a hard, solid mass.		Tumour or tumours to be distinctly appreciated; possible mobile, and feel cystic and fluctuating.
Percussion.	Dulness, particularly in the flanks with patient on back, possibly only here appreciable, and definitely shifts by *imperceptible* movement as the patient shifts his position.		Dulness usually only appreciable over the tumour, not *specially* in flanks; if it shifts with position of patient there is a *perceptible* movement of something inside the abdomen, detectable by palpation also.
Thrill.	Frequently to be detected through the whole abdomen.		Usually not obtainable at all; if it is so, it will only be in a limited area through the body of the cyst, not through the whole abdomen.
Other means.	In females bimanual examination gives no very distinctive information.		Bimanual in a female shows movements of uterus with ovarian cyst; other cystic formations may be felt definitely as such, of limited extent or mobility, or both. In ovarian cyst menstrual irregularities may also help us.

The only case which is likely to cause much trouble is that of a large, thin-walled ovarian cyst, which has practically filled the whole abdomen. Here even a careful attention to the above points may still leave us in doubt. The history of menstrual irregularities, and of a previous asymmetry, together with an absence of all presumable

causes of ascites, will probable put us right; such cases are nowadays very rare, and, of course, they can only occur in the female sex. Very great obesity of the abdominal wall is a great obstacle to physical examination, and such a case demands extra care, with which difficulties may vanish, but frequently only an examination under a general anæsthetic will clear up a doubtful abdominal case.

Simple Dyspepsia *v.* Gastric Ulcer

Even in an elementary work dealing with diagnosis it seems impossible to omit all reference to this subject, for the cases are so common in which we desire to have guidance, and yet the definite separation of the two conditions—perhaps it would be more correct to say the exclusion of ulcer—is so extraordinarily difficult, that I feel it is much easier to criticise the criteria mentioned in text-books than to offer any distinction of my own.

If we take a number of cases of gastric ulcer, either proved such by autopsy or diagnosed as such during life, we find the following points more or less marked :—

1. The subjects will roughly divide themselves into two groups: (*a*) those whose ages range from seventeen to thirty; (*b*) those from forty onwards. Group (*a*) will be almost entirely composed of the female sex, and the majority will have suffered from anæmia, and they will not have wasted. Group (*b*) will be composed of either sex indifferently: anæmia—except for hæmatemesis—will not have been marked, but loss of flesh will commonly have appeared.

2. Severe pain will have been a prominent feature in the history of the illness, and this pain will have frequently displayed the following peculiarities :—

(*a*) Caused only by taking of food.
(*b*) Appeared some little time after the ingestion of food, *not at once.*
(*c*) Localised to a limited area, usually in the epigastric angle.
(*d*) Increased by pressure on the spot, and often only then apparent.
(*e*) Relieved almost immediately by vomiting when this has occurred.

3. Vomiting is also likely to have been frequent, with chief characteristics :—

(*a*) It occurred rather late after food.

(*b*) It relieved the pain almost at once.
(*c*) It has been actual blood or blood-tinged at least once.

4. In the cases fatal from ulcer there will almost invariably have been, in addition to the above:—

(*a*) Perforation with acute peritonitis; or
(*b*) Very severe hæmatemesis; or at least
(*c*) Such constant severe pain and vomiting as to have led to death by exhaustion—this last mode of death is commoner in the group of older patients.

Hence with regard to diagnosis it is very easy to say that an ulcer is present when the above symptoms occur in typical severity. It is the converse of this proposition, viz. to exclude an ulcer when gastric symptoms are slight, that offers such enormous difficulties; in fact, there can be no hesitation in saying that it is *impossible by clinical methods alone to prove this negative.* Will the Röntgen rays here again help us?

It is being constantly proved in cases that are operated upon for perforation, less commonly in ulcers found on autopsy, that pain may be—

(*a*) So slight as to be practically unnoticed.
(*b*) Relieved by taking food.
(*c*) Generally diffused and not localised.
(*d*) Unrelieved, or only very gradually so by vomiting.

That vomiting may be—

(*a*) Absent altogether.
(*b*) Not bloody nor blood-stained.

In fact, that in certain cases every one of the guiding indications may have been absent, or what is perhaps worse, may have been absolutely contra-indicative. These exceptional cases are far more common in the group of young subjects; in the older group the features are more constant, though not even there entirely without exceptions.

Dr. S. Fenwick has adopted, if not invented, a simple method of attempting to solve the diagnosis by getting the patient to swallow about a drachm of common salt in a tumbler of water when the stomach is empty; should an ulcer be present, the salt coming in contact with a raw surface will excite a lively attack of pain; if ulcer be not present, pain is not likely to be caused by the simple procedure. The same plan may be adopted to determine whether an ulcer, which has been diagnosed as present, has healed.

Apart from this clinical manœuvre we can, I think, only lay down the following rules :—

1. Never to think too lightly of an attack of dyspepsia in a young woman, especially if she be anæmic as well.

2. Whenever it is possible, even at some inconvenience to the patient, to insist on a soft diet and a few days' rest in bed in all cases of dyspepsia which have not arisen from a fairly definite cause.

3. A persistent-local (epigastric usually) pain in an older patient must similarly put us on our guard, to insist upon treatment appropriate to ulceration.

By attention to these rules we may cause some trouble to ourselves and our patients, but we shall at least have the satisfaction of having avoided anything likely to make matters worse.

CHAPTER VI

DISEASES OF THE URINARY ORGANS

One of the most striking facts about kidney troubles is the frequency with which they occur without local symptoms. In acute nephritis it is true that aching pain in the loin is frequently present, and new growths of the kidney, including stone under that head, are usually associated at some period of their growth with more or less severe local pain: but chronic Bright's disease, lardaceous degeneration of the kidney, and, in fact, the majority of those cases in which the most serious degeneration and disintegration of the kidney structure are present, only betray themselves by general symptoms due to imperfect depuration of the blood, and by alterations in the urine of such a character, or so insidious in onset, that they may not have attracted the attention of the patient.

For a discussion of the general symptoms refer to the heading Uræmia. The alterations in the urine are sometimes sufficiently obtrusive to be called symptoms, sometimes so insidious that they must be relegated to the class of physical signs, but in either instance they are so exceedingly important that they must be rather fully discussed for diagnostic purposes.

Before we can appreciate or estimate *pathological* changes in the urine we ought to have a satisfactory general idea of the quantity and quality of the *healthy* excretion, and also of its physiological or healthy variations—a subject, which long experience of students has taught me, is dreadfully neglected, and forgotten as soon as the examination in physiology is passed.

Normal Urine

Should possess the following characteristics:—

Quantity.	From forty to fifty ounces in the adult.
Appearance.	Clear and bright.

Colour.	Amber to sherry, light or dark.
Reaction.	Acid.
Sp. gravity.	1015 to 1025.

It should, in an adult, be passed about four or five times in the twenty-four hours, and should on analysis show about 2 per cent of urea.

The colour is due to various urinary pigments, ultimately, no doubt, derived from bile pigments and from hæmoglobin.

The reaction is due to the presence in fairly large quantities of the acid phosphate of sodium NaH_2PO_4 (*and probably of the analogous potassium salt also in smaller quantities*). It is truly wonderful how this simple, well-established fact is forgotten by students, who will persist in ascribing the acidity to uric and hippuric acids, regardless of the feeble acidity of these bodies, and their insignificance in quantity.

Variations in Health

In health the following are the chief factors influencing the urine, and a brief epitome of how and why.

1. *Exercise.*—Physical work must undoubtedly—by mere wear and tear of fixed tissues as well as by quickening circulation, and therefore oxidation of more floating capital—have a tendency to throw more waste products into the blood. These waste materials are the ultimate oxidation products of organic food stuffs, H_2O, CO_2, and urea or uric acid (as urates); the H_2O and CO_2 pass off in large measure through the lungs by increased respiratory movement, and through the skin with the sweat, which is also markedly increased by exercise; hence there follows as a natural deduction, just what is seen in actual practice, viz. that with exercise the urine shall as a rule be lessened in quantity, but increased in specific gravity, by a higher proportion of nitrogenous waste material and inorganic salts.

At first this nitrogenous waste takes the form of uric acid, so that a copious deposit of urates and biurates is the familiar result of a hard day's work in one who is unaccustomed to physical labour. As uric acid is a less oxidised product than urea, *i.e.* contains a smaller proportion of oxygen, it is probable that this deposit is due in some way to an inefficient supply of oxygen to burn up the increased waste suddenly produced. When the body has had time to accommodate itself to the circumstances, the more usual metabolism of nitrogenous waste into urea again comes to the fore.

2. *Food.*—The ingestion of food influences the urine in two ways:—

A. Directly: by the simple excretion of excess of water taken in—the student is apt to forget that the so-called solids of the food (*a*) contain a large proportion of free water, (*b*) produce much water by their oxidation, so that it is not only the drink alone of the food, but the solids also that produce this excess.

B. Indirectly: during gastric digestion a quantity of HCl is required, the manufacture of which sets free a corresponding quantity of sodium in the blood, with the result that the phosphate of sodium in the urine is more likely to become Na_2HPO_4, or even Na_3PO_4, rather than NaH_2PO_4.

By both these processes, then, the quantity, the specific gravity, and the acidity of the urine are likely to be influenced—the first increased, and the last two diminished; the acidity to such a degree that it is quite within physiological limits to find the urine neutral or even alkaline after a full meal.

3. *Weather.*—This simply has reference to the temperature to which the skin is exposed when no compensating influences—such as exercise—are at work on the circulation through it. Cold causes the skin to contract, and diminishes consequently its circulation and excretion of sweat, with the result that the amount of urine passed is increased; external heat will have the contrary effect.

4. *Mental Conditions*, such as fear, certain hysterical states on the borderland of health, and especially doubt as to opportunities for voluntary micturition, undoubtedly have an influence on the secretion of urine; but their exact mechanism is obscure, and we can only record the fact.

Thus we see that—

Quality, Sp. gr., Reaction, } May all be profoundly altered by exercise, food, weather, and mental conditions.

We will now proceed to discuss from a diagnostic point of view the variations in urine due to disease.

Variations in Disease

Increased Frequency of Micturition

May to due to—	Chief diagnostic points.
1. Acute inflammation or irritation of kidney.	Very small quantities at a time, total quantity much diminished, probably blood in it; strangury not infrequent.
2. Stone, or other growths of kidney, with colic.	Also like acute inflammation, but with severe pain (renal colic, *q.v.*) probably running down into groin.
3. Cystitis and bladder calculus.	Total quantity probably about normal, possibly alkaline and ammoniacal, certainly much mucus deposit; probably pain in bladder or perineum, which may only be at end of act, and often referred to the glans penis.
4. Prostatic troubles.	If acute the act is itself very painful; if chronic the increased frequency of micturition is chiefly at night, with possibly some difficulty in starting the act during the day, and the catheter (possibly passed with difficulty) shows residual urine; total quantity normal or greater than usual.
5. Polyuria.	*Vide* next section; total quantity much increased.

Total Quantity

Is Diminished in

1. Acute congestion or inflammation, if primary.	Frequent micturition; strangury; smoky or definitely bloody urine; albumen and casts; possible history of turpentine or copaiba, etc., administration.
If on the top of old mischief.	The same features, but with history of previous increased quantity.
2. Large white kidney.	*Vide* section on L.W.K. Urine nearly solid with albumen; patient very anæmic.
3. Blocking of ureter.	If of both, total suppression (*q.v.*); if one only, such urine as escapes possibly normal; probably severe pain in groin and loin.
4. Retention, heart disease, and general circulatory disturbances.	*Vide* Suppression *v.* Retention, p. 216. Moderate quantity of albumen; history of *gradual diminution* in quantity of urine, and presence of other signs of heart disease or circulatory disturbances.

		Is Increased in
A.—Affections of kidney.	Cirrhotic kidney.	Low specific gravity, little albumen, patient probably over forty-five years of age; few if any casts.
	Consecutive nephritis.	Probably considerable number of casts; fair quantity (up to one quarter) albumen, and history of acute kidney trouble.
	Lardaceous kidney.	Probably also enlarged spleen and liver, and known cause of lardaceous degeneration present; albumen considerable, casts may or may not be numerous, and possibly show lardaceous microscopic reaction.
	Hydronephrosis.	Increased quantity only at irregular intervals, and urine quite likely without much change from normal; abdominal tumour in renal region often detectable.
B.—General Conditions.	Diabetes insipidus.	Quantity enormous, up to 300 or 400 ozs.; specific gravity very low; absence of albumen or other pathological constituent.
	Diabetes mellitus.	Specific gravity usually above 1025, *but, N.B.—sugar may be present in urine of specific gravity below* 1020; presence of sugar; urine of peculiar appearance and general symptoms of diabetes.
	Hysteria, after rigors of malaria, occasionally after crisis of acute disease.	Urine offers no special peculiarities; the polyuria is quite temporary, and the attendant circumstances are quite sufficient for diagnosis.

Colour

Of pathological variations in colour due to changes in the urinary pigments very little is known, and still less use can be made for clinical purposes of the knowledge we do possess (perhaps with the exceptions of melanin and indican); but the following substances when present will produce evident colour variations from the ordinary, and these variations form the first preliminary or simple test for the respective substances, and should therefore be remembered:—

Bile pigments.	Urine a yellowish green tinge in thin layers, almost black in thicker ones; if shaken the froth possesses a beautiful yellow colour. This yellowness of the froth is pathognomonic of bile in urine (? rhubarb or santonin).
Blood.	Urine either of a smoky tinge and not clear, or possesses the red colour of blood more or less marked.
Chyle.	Urine distinctly milky throughout, and often coagulates into a soft jelly.
Carbolic acid.	Urine of a green colour, looking almost black in thick layers.
Rhubarb.	Brownish yellow colour, becoming red on adding alkali, disappearing with an acid.
Santonin.	Bright yellow; disappears with free acid.
Hæmatoxylin.	Reddish, also disappears with free acid, thus differing from red colour due to blood; *vide* Hæmaturia.
Iodides and bromides.	Dark colour appears on adding nitric acid, due to setting free of iodine or bromine.
Methylene blue (*occasionally given for rheumatoid-arthritis*).	Urine of a bright blue colour. I have recently seen a case of eosin in the urine mistaken for blood—it arose from eating sweets coloured with eosin.

Specific Gravity

Apart from the knowledge of the total quantity passed in twenty-four hours, variations in specific gravity have very little positive pathological significance. A high specific gravity (over 1027) should, however, at once rouse a suspicion of glycosuria (*q.v.*), while a low one (under 1015) should immediately start inquiry into the total quantity passed, etc., leading in the direction of suspected cirrhotic kidney, unless other obvious explanation be at hand.

Constituents of Urine not usually found in Health, and essentially Pathological

By their presence.	Blood, pus, casts, sugar, albumen, chyle, phosphate of lime if in quantity, bile.
Or by excess.	Urates, mucus.
Or by altered conditions of urine.	Triple phosphates, ammonia.

For convenience of discussion, and because of its practical utility in urine analysis, we may divide these constituents into those

which form, or are chiefly found in association with, a deposit of some sort, and those which still leave the urine quite clear notwithstanding their presence, thus :—

Forming, or chiefly found in, a Deposit.	Leaving the Urine clear.
Phosphates of lime.	Sugar.
,, triple.	Albumen.
Urates and uric acid.	Blood. (If in quantity usually makes urine opaque.)
Oxalates.	Chyle. (If in quantity usually makes urine opaque.)
Mucus.	Bile. (If in quantity usually makes urine opaque.)
Pus.	
Casts.	
Blood staining a precipitate.	

It is obvious that there are two questions requiring answers in connection with these abnormal constituents—

(1) By what tests shall we recognise them ?
(2) What clinical diagnostic use can we make of the information thus acquired ?

To the first of these questions I propose to give merely an outline of a reply; for details of manipulation special manuals must be consulted, as I am chiefly concerned with clinical and not scientific questions.

Tests for the Nature of an Abnormal Constituent of the Urine

There are three methods to be adopted in testing urine, all of which should be employed in turn as corroborating one another: (1) ordinary physical examination by the eye and nose; (2) chemical experiments; (3) microscopical investigation; if very great accuracy is demanded, the spectroscope may be used to detect blood and bile.

Smell

Of ammonia.	Indicates decomposition, and so far gives a clue to the improbability of a deposit being urates, but suggests pus or triple phosphates.
Of violets.	That the patient has been taking turpentine.

These are the only two indications that smell is likely to give one, but asparagus and possibly other articles of diet do alter the

natural smell of healthy urine, though the fact is not of much clinical value.

Appearance to Naked Eye of Urinary Deposit or Urine

Phosphate of lime.[1]	White, more or less granular deposit; sometimes in large amount (= phosphatic diabetes), and may be hummocky from intermixture of mucus.
Triple phosphates.[2]	Only recognised accurately by microscope in a deposit; urine ammoniacal.
Urates.	Usually coloured pink or red; if white they may be distinguished from phosphates by the fact that they usually adhere to the side of the vessel at the top of the urine.
Uric acid.	Only seen as isolated little cayenne-pepper grains at the bottom of the glass, or entangled in mucus.
Oxalates.	Usually seen (if at all by naked eye) as small shining crystals entangled in the mucus.
Mucus.	Practically a constant feature even in health; it forms a translucent, semi-transparent, flocculent cloud towards the lower part of the glass, and often floating free in the middle of the urine.
Pus.	White or greenish white, often hummocky deposit at the bottom of the glass, not granular, but easily mistaken for phosphates or white urates; mucus cloud usually excessive above it; urine commonly, though by no means invariably, ammoniacal.
Blood.	Smoky or definite red colour of blood, light or dark.
Sugar.	Peculiar greenish yellow tinge in typical diabetic urine, but perhaps more often nothing very distinctive.
Casts and Albumen.	Only observable under the microscope; not any characteristic appearance.
Bile.	*Vide* colour and chemical changes, p. 190

[1] Students are very apt to forget that phosphates of sodium and potassium are soluble in water, and therefore do not occur in precipitates.

[2] These crystals are called triple phosphates, because all three atoms of hydrogen are replaced by metals (2 by Mg, 1 by Am); they can only occur when decomposition has taken place in the urine, either within or without the body; for without decomposition ammonia does not appear in the urine.

Heat and Chemical Tests

First ascertain by litmus paper the reaction of the urine. Inasmuch as all the deposits may occur in acid or alkaline urine, with the exception of triple phosphates (*vide* footnote, pp. 192 and 209) and excessive urates, an alkaline reaction gives no very definite information beyond a strong probability that a deposit is not urates, but it is important in the heat test for albumen.[1]

Then take a little of the urine in a clean [2] test-tube (if there be a deposit to be tested take care to get plenty of it in the test-tube), and apply heat very cautiously and gradually, watching the urine carefully all the time. Bring it to active ebullition, and finally add a drop of nitric or, better still, acetic acid.

The meaning of this is as follows:—

1. From cold up to, say, a little below blood heat urates will gradually dissolve:

∴ Watch the deposit to see if it gets lighter—the diminution represents urates; if no diminution up to this point, no urates; and, *N.B.*—all urates are now dissolved, and so cannot be mistaken for albumen.

2. From about blood heat up to boiling albumen will, if present, be coagulated and appear as a precipitate; the more albuminous a liquid the lower the temperature at which the albumen will *begin* to coagulate:

∴ Watch the deposit (or clear urine) to see if it gets thicker (or clouds).

Any precipitate now appearing is either albumen or phosphates,[3] and neither will disappear on boiling, so we add one drop of acid (acetic is best, as it is weaker and so avoids fallacies of nitric dissolving traces of albumen), which—

3. Dissolves any phosphates, but (if not too much be added) leaves the coagulated albumen untouched:

[1] This is owing to the fact that when decomposition takes place any albumen present is converted by the ammonia into alkali-albumen, which is not coagulated by heat alone.

[2] Dirty test-tubes, especially with acid, will often prevent precipitation of albumen.

[3] It is doubtful to what cause this deposition of phosphates is due; possibly escape of CO_2 which held them in solution, or possibly some subtler chemical change in them produced by boiling (Ralfe).

∴ The part of final precipitate clearing up with acid = phosphates; remainder = albumen or other proteid body; if brownish red, probably due to blood or stained by blood.

4. If the deposit is unaltered or only slightly thickened by boiling, it is either phosphates or pus.

Now take a fresh specimen in a clean test-tube, and add a drop or two of acetic acid or weak nitric acid, this—

Makes mucin stringy;
Dissolves phosphates;
Does not affect urates;
Does not materially affect pus.

To a similar specimen add a few drops of caustic potash; this—

Causes pus to become jellified or ropy;
Dissolves urates;
Does not affect urates markedly.

To test for sugar, boil some Fehling's solution, and while boiling hot add a few drops of urine; if sugar be present, red or yellow oxide of copper is rapidly precipitated. There are many other tests for sugar, and great controversy still exists as to which is the best; but the above, known as Fehling's test, is sufficient and accurate enough for ordinary clinical purposes.

If bile be suspected by the colour, Pettenkofer's test with fuming nitric acid, giving a play of colours if bile pigments be present, may be applied as confirmatory evidence. But the simplest of all tests for bile is the yellow colour imparted to the froth that appears when urine is shaken.

Microscopical appearances of Urinary Deposits and Elements

Oxalates.	
Phosphates—	Amorphous or crystalline.
Triple phosphates.	
Pus.	
Blood cells.	
Urates.	
Uric acid.	
Casts.	

CLINICAL SIGNIFICATION OF ABOVE

We may now proceed to discuss more fully the second question mentioned above, viz. What is the clinical significance of the various morbid constituents, and how far are they of diagnostic value?

Albuminuria

The causes that may produce albumen in the urine are many, but we can distinguish in clinical work three groups of albuminuria :—

Group I.—In which the albumen is only a subsidiary factor, and its presence completely accounted for by the other constituent, viz.—	Blood (*vide* Hæmaturia). Pus (*vide* Pyuria). Spermatorrhœa (?).
Group II.—Present with other factors explaining its anatomical source, viz.—	With abundant casts, indicating acute kidney disease, or consecutive nephritis in earlier stages.
Group III. — In which it is often the only obvious morbid constituent, though a few casts or crystalline substances may be present in some cases.	Cirrhotic kidney. Consecutive nephritis in later stages. Lardaceous kidney. Surgical kidney (hydro-nephrosis). Stone in pelvis, probably with pus or crystals. Cardiac disease or general back pressure. Abdominal disease or pregnancy, with local back pressure. With or after some specific fevers—diphtheria, scarlet fever, etc. So-called cyclical or functional albuminuria. Leucorrhea in women. Anæmia (occasionally). Debility in or after many general diseases.

To determine to which of these groups a given case belongs it is obvious, then, that we must examine the urine carefully (and repeatedly in some cases) by chemical and microscopical[1] tests.

[1] If no obvious deposit is present let the urine stand for twelve hours in a conical vessel covered from dust. The late Dr. Ralfe used also to dust a little powdered starch on its surface, to carry down mechanically any floating casts. If a centrifugalising machine is available this is the best method of obtaining a deposit.

These will be practically certain to differentiate Groups I. and II., and by exclusion lead us to believe that some member of Group III. is causing the mischief.

Then note the quantity of albumen present; if only a trace, it is most likely due to—

Cirrhotic kidney.
Back venous pressure (local or general).
Surgical kidney (first or second stage, *q.v.*).
Specific fevers (not reaching stage of actual nephritis).
Leucorrhea.
Anæmia.
Cyclical or functional (*q.v.*).

If more than a trace, probably—

Lardaceous kidney, or
Consecutive nephritis without many casts, or
Chyluria.

Most of these conditions will become tolerably obvious when the patient as well as his urine is carefully overhauled, but one or two of them require a little further consideration.

Cyclical or Functional Albuminuria

These are terms invented to describe cases which were, I believe, first noticed by examiners for life assurance (but the condition has subsequently been investigated by many observers), who found albumen in the urine without obvious cause, and with an entire absence of other guiding pathological constituent or even symptom. Its exact pathology, and above all its precise clinical significance from a prognostic point of view, are still but imperfectly understood, but before we can allow ourselves to rest content with such a diagnosis, the following propositions and conditions must have been rigorously investigated :—

1. That the patient must have been most carefully examined in every system, without finding evidence of disease (more especially, perhaps, the condition of the vascular system).

2. That the urine and the urinary system must have been also *repeatedly and carefully* examined without the discovery of any morbid constituent or local condition, especially casts, blood, pus, stricture, stone, phimosis, leucorrhea, fever, phthisis, leucocythæmia, etc.

3. That the albumen shall never have been more than a trace.

4. It is a condition hitherto found almost exclusively in adolescents (boys or girls), or youngish adults, though it is uncertain how far it may persist into later life.

5. The circumstances found to influence the presence and quantity of the albumen have usually been position of body, exercise, exposure, and certain articles of diet, *e.g.* eggs; and hence the urine must be examined on several occasions—

Before and after rising in the morning;
" " cold bathing;
" " food of suspected kinds;
" " exertion (slight and severe).

If these various testings give various results while other conditions remain the same, a preliminary diagnosis of functional may be given, but one to be willingly abandoned on the discovery of some indisputably organic change.

Surgical Kidney as Cause of Albuminuria

Surgical kidney is a convenient, but somewhat invidious, expression used to denote a kidney which has been damaged by obstruction to the free outflow of urine from its pelvis. It would appear that an intermittent obstruction is equally, if not more, efficacious than a permanent one in producing the condition.

In its typical form it is essentially a surgical trouble with surgical treatment, and its description and discussion must be looked for in surgical manuals; but as its earlier manifestations and irregular forms may easily come before a physician, its main features are here glanced at.

There are three forms or stages of it readily distinguishable when met with alone, but they are far more usually—on the post-mortem table at any rate—mixed with one another:—

1. Simple atrophy of pyramids from pressure.
2. No. 1, plus chronic inflammation of kidney from irritation.
3. No. 1 and No. 2, plus septic infection of the urinary tract.

As regards the causation, there are three well-known causes, viz. stricture of urethra, stone in the bladder, and enlarged prostate, and when either of these is in operation it is probable that both kidneys will suffer. The disease is here mentioned in a medical work chiefly on account of a group of cases which arise from very

ill-understood causes that are supposed to give rise to intermittent kinking of the ureter, with a consequent intermittent increase of pressure in the pelvis.

Definite diagnosis of such cases is in all probability impossible in their earlier stages, but the following symptoms would render the condition highly probable :—

1. Albuminuria probably not more than a trace, and very possibly intermittent.
2. Marked irregularity in the quantity of urine passed in successive days.
3. The presence in the abdomen of a tumour, possibly ill-defined, diminishing and increasing according as the amount of urine passed is large or small.

The remaining conditions of albuminuria in Group III. may be more briefly dismissed in tabular form :—

Condition.	Guiding Indications.
1. *Cirrhotic kidney, or consecutive nephritis in the later stages.*	Specific gravity low ; quantity increased ; heart hypertrophied ; vessels probably degenerate ; distinct history of nephritis in consecutive cases.
2. *Lardaceous kidney.*	Albumen considerable ; quantity of urine greater than usual ; some obvious cause for lardaceous change, not forgetting syphilis and phthisis ; liver or spleen, or both, almost sure to be enlarged ; painless diarrhœa may (rarely) be present.
3. *Heart disease.*	Bruits probably present, or at least alterations from health in the cardiac sounds ; nearly sure to be œdema of legs ; quantity of urine *diminishing lately, pari passu* with the onset of other back pressure symptoms.
4. *Abdominal disease.*	Ascites or tumour or pregnant uterus ; swelling of abdomen probably obvious.
5. *Nephrolithiasis.*	*Vide* Stone in Kidney.
6. *Diphtheria, scarlet fever, or other zymotic; anæmia, leucorrhea, convalescence from even acute nephritis.*	The history of such disease is usually obvious enough ; albumen likely to diminish as convalescence proceeds, but casts must be anxiously looked for.

In connection with all the groups, but especially perhaps with

this last group, of causes of albuminuria, a very anxious question will often arise—Is this patient weak and anæmic because he is albuminuric, or is he albuminuric because he is weak? The question is important because of treatment. If we feed him up to cure anæmia we may further damage his kidneys: if we starve him to relieve the kidneys we may perpetuate the anæmia, and also the debility of renal tissue. To answer this question fully would require an essay, but the following points are worthy of attention:—

It is obvious at once that all albuminuria depends upon and is caused by—

(1) Acute or gross disease in or near
(2) Simple nutritive (or original) debility of

some part of the kidney structures, which, when healthy, prevents or does not cause a transudation of albumen into the excretion.

Acute or gross disease includes—

(*a*) All those cases (stone, tubercle, carcinoma, perirenal abscess, etc.), the treatment of which is or should be almost entirely surgical, and of which a brief note will be found under the appropriate heading.

(*b*) Inflammation of the kidneys or bladder, of which, when acute, the treatment and management are obvious enough.

Difficulties and doubts arise when the acute trouble has subsided and the case become one in which the albumen remains (with the anæmia in the secondary cases, possibly without it in the primary ones) as practically the only sign of disease. These must be placed for our present object along with the group in which no such acute local trouble has been present. The following important propositions may then be made on them, each proposition having weighty therapeutical deductions:—

1. Albuminuria of so-called functional or cyclical origin may exist for years without the subject of it showing any marked deterioration of health, and the same is true of many albuminurias which are the sequel of known acute Bright's or other gross renal mischief.

2. If the urine on boiling becomes practically solid, this only means about 2 per cent of albumen—say the equivalent of an egg or a couple of ounces of beefsteak in twenty-four hours; while what is ordinarily spoken of as a trace of albumen means from $\frac{1}{2}$ to $\frac{1}{10}$ per cent or less—the equivalent of about one ordinary mouth-

ful of food—so that as a mere drain of nutrient material from the body albuminuria may be certainly neglected.

3. Experience has shown, after many trials, that no drug and no plan of low diet expressly used, *ad hoc*, has hitherto been found capable of preventing or eliminating the last traces of albumen from the urine.

4. It is the life-work of the kidneys to excrete some of the waste products from the body, and these oxidation products come from two sources:—

(*a*) The result of the ordinary never-ceasing activity (life, growth, and decay) of all the body tissues and cells.

(*b*) A possible, or even probable, luxus consumption of materials taken in by the mouth as food, but never really assimilated into the structures of the body.

Now, any rational system of management of albuminuria must take all these facts into account. Wear and tear of tissues cannot be checked entirely, but it may be kept within moderate limits by avoiding excessive physical exertion. Luxus consumption can also probably not be entirely avoided, but may be reduced considerably by *avoiding excess of* nitrogenous food. Beyond these extremes I do not think we should be too strict either in diet or exercise with albuminurias, for constant worry of obeying rules is worse than occasional lapses from them.

Pyuria or Pus in the Urine

The sources of pus found in the urine may thus be tabulated, with their most prominent guiding features:—

Source.	Chief Points.
Urethra, in either sex.	Quantity small; escapes at any time, independently of micturition, and stains and stiffens the linen; micturition probably painful; squeezing urethra towards meatus shows a bead of pus.
Vagina in female.	Similar indications, and speculum clinches the diagnosis; also vaginal epithelium revealed by microscope.
Bladder.	Most probably associated with frequent distressing micturition and other symptoms of cystitis; urine possibly ammoniacal on voidance; much bladder epithelium under microscope; if no symptoms (or very slight) of cystitis, pus prob-

Source.	Chief Points.
	ably in small quantity, but the cystoscope must be appealed to as final arbitrator.
Ureter.	Practically indistinguishable from kidney.
Kidney and pelvis thereof.	Quantity usually considerable; stone and tubercle most common causes; urine most commonly acid, and but little disturbance in frequency of micturition, except in stone; pain of a colicky nature not unfrequent; renal epithelium frequently found under the microscope.

If we have ascertained the place of origin of the pus, the next point is the cause of the suppuration itself:—

1. *In urethra.*	Hunterian chancres have been known to occur in the urethra; the finger would readily detect them; instrumental irritation. Strain has lately been asserted on good authority to cause a urethritis independently of sexual connection. Gonorrhœa, recent or old (with stricture), is, after all, far and away the commonest cause. If the matter is legally disputed the gonococcus would be the only possible distinctive feature, though this is more likely to lead to hard swearing than to scientific conviction.
2. *In vulva and vagina.*	Such causes as foreign bodies (pessaries, etc.) are obvious on inspection with a speculum, as are also chancres, epithelioma, etc. It is indisputably proved that in young subjects dirt, masturbation, etc., may start a suppurative inflammation without a suspicion of criminal violence. A diagnosis without a history is impossible, but the fact of such vulvitis occurring must be remembered by a medical jurist.
3. *In Bladder.*	That suppuration may occur in the bladder, or at least that pus may enter the urinary tract at the bladder without general cystitis, is true; but its occurrence is so rare that practically the causes of cystitis and of suppuration through or from the bladder are the same. They are:—

(*a*) Foreign bodies.	Stone.—Proved by the sound.
	Fæces or septic pus.—History of illness, such as typhoid, parametritis, pyosalpinx, which could give rise to entero-cystic fistula or abscess bursting into the bladder.
	Bits of catheter, etc.—History generally admitted, but may be only recognised by the sound.
(*b*) Distension from	Stricture of urethra.—Proved by the catheter and history of difficult micturition.
	Enlarged prostate.—Found by catheter or finger in rectum.
	Simple atony. — Residual urine drawn off by catheter.
	Uterine enlargement or puerperium ; obvious.
(*c*) Extension of inflammation.	History of acute urethritis or vaginitis, possibly diphtheritic.
(*d*) Debility.	Cystitis arises in the very old or very young, or in patients debilitated by any illness, from very trivial causes, entirely overlooked perhaps, and not operative in health, *e.g.* clean coitus, clean catheter.
(*e*) Ulcers—Simple, tubercular, malignant, primary, or extending to bladder from other organs, especially uterus.	The tubercle bacillus or fragments of a neoplasm *if present* will, of course, settle the matter for two of these, but otherwise to be certain of them is impossible without the aid of the cystoscope.
4. *From kidneys and ureters.*	The causes of suppuration from kidneys and ureters are not very numerous.

Principal Points

Tubercle.	Pus abundant (may be none, *vide* Tubercle of Kidney), and hæmorrhage, too, not unfrequent ; tubercle bacilli in pus ; renal colic not unfrequent ; possibly tubercle elsewhere. Age : middle life usually.
Stone.	Pus as a rule not very abundant ; colic common (*vide* Nephrolithiasis). Age : children and old people very prone to it, but no age exempt.

Malignant disease.	Pus not much; hæmorrhage more likely (*vide* Kidney Cancer). Age: late adult life, except rapidly growing sarcomata, which are almost confined to children.
Pyæmia.	Either extension from the bladder, when the cystitis will mask the kidney symptoms, or a cause of general pyæmia present; in either case renal features quite subordinate.
Perinephritic abscess.	History of increasing pain and swelling in the loin; pus in urine increasing or diminishing with diminution or increase in tumour, and often quite intermittent.

In all the above renal troubles if pain or tenderness be marked on one side, and not on the other, or if a very distinct tumour be felt by abdominal examination, the kidney at fault will easily be known; but it is a very common thing for the diagnosis to remain in doubt until the cystoscope has been used, showing something abnormal in or of the ureteral orifice of the affected kidney.

Hæmaturia

Blood like pus may come from any part of the urinary tract, and in the main the indications of its source are similar.

Chief Indications

Urethra.	Is pure, and escapes at any time, thus staining the clothes; probable history of traumatism or instrumentation.
Prostate.	Blood from prostate either gets into the bladder, or trickles down the urethra; in either case the prostate would only be suspected if other symptoms of enlarged prostate had occurred, or if digital or instrumental examination had revealed prostatic abnormalities.

Table of Differences of Hæmaturia from

Bladder.	Kidneys.
Usually very obvious blood; often irregular clots in it; blood frequently by itself.	More commonly a smokiness, not obviously blood, more intimately mixed with urine; if clots, they will have shape of ureter.

Bladder.	Kidneys.
No renal casts; possibly bladder epithelium.	If from renal substance, almost invariably blood casts of renal tubules.
Strangury[1] a less common association.	Strangury a common association, *e.g.* after taking turpentine or application of cantharides.
If any local pain it is in bladder region, or at end of penis.	If any local pain it is in renal region, or in groin and testes.
Never hæmoglobinuria only.	May be only hæmoglobinuria, *i.e.* no evident corpuscles.

Notwithstanding these indications, the cystoscope will frequently have to be used to clear up the diagnosis.

Tubercle bacilli or bits of malignant growth may occur in either, and offers no assistance in localising diagnosis.

If we decide that the blood comes from the kidney, we have the following points:—

From Hilum.	From Substance.
Free hæmorrhage and ureteral casts.	Smokiness merely, and casts of renal tubules; if hæmorrhage is free traumatic history almost invariable.
Never hæmoglobinuria.	Often hæmoglobinuria only.
Frequently associated with pus.	Practically never with pus, the only exception being pyæmia.

As with pus, we must consider the diagnosis of the cause of the hæmorrhage.

1. From urethra, vagina, and vulva.	Traumatism, carcinoma, etc.; the parts are so open to inspection that nothing further need be said.
2. From prostate—	
Carcinoma.	Enlargement revealed to examining finger in rectum; chronic history of local pain.
Abscess.	Tenderness to finger; possibly a fluctuating swelling; acute history of prostatic trouble; pus with the blood.
Varicose veins.	These unquestionably exist on an enlarged prostate and at the base of bladder, and may

[1] "Strangury" is a term used to express a pathologically excessive desire to pass water, with frequent attempts to fulfil the desire: it is a symptom of excessive irritation of kidneys, and probably reflexly of the prostate too.

give rise to severe hæmorrhage; their presence will chiefly be guessed at by the absence of other causes; only the cystoscope can *prove* their presence.

3. Bladder. The causes of hæmorrhage from the bladder are the same as those of pus (*q.v.*) with the exception of simple varicose veins, and innocent papillomata. In the late stages the diagnosis will be self-evident; in the earlier stages the cystoscope or sound is simply and absolutely indispensable; without one or possibly both of them diagnosis is impossible.

4. Kidney. Unless one of the following—heart disease (for infarcts), tubercle bacilli, fragments or new growth, or tumour in loin—is present, and reveals by its presence or by its character the nature of the kidney trouble, we may say at once that exact diagnosis of the cause of renal hæmorrhage is impossible without exploration. Acute nephritis (with its attendant general symptoms), hæmophilia, and those cases in which only some form of blood pigment (without corpuscles) is present, are of course excepted; their diagnosis being more from general considerations than through the local hæmorrhage. I have seen very obstinate renal hæmorrhage follow influenza without anything occurring to give any hint of a more definite diagnosis. Stone, tubercle, and carcinoma are far the most frequent, and must be suspected until other cause can be found or definitely excluded.

CASTS

To mention all casts possibly found in urine it must be stated that urethral or ureteral casts may be met with; they will, however, usually be recognisable by the naked eye, and will be simply blood clots. They have been sufficiently noticed under Hæmaturia.

It remains, therefore, to enumerate the various kinds of renal microscopic casts that are met with, and consider their value as diagnostic factors. As a convenient method of remembering them,

we may put them down in the order in which they must occur in a case of acute nephritis from the stage of congestion onwards. Thus we shall have—

1. Small hyaline casts from simple coagulated serum exuded in the early hours of congestion, generally overlooked owing to the rapid production of—
2. Blood casts, which form from the blood escaping into the uriniferous tubules in the next stage of the inflammatory process; inflammation rapidly causes the death of renal epithelium, and consequently—
3. Epithelial casts soon follow, formed of masses of rapidly-shed epithelium. That which at first remains attached to its membrane, but eventually dies, undergoes degeneration, and disappears from the tubules as—
4. Granular casts,—if the cells degenerate so as to lose their outline, and become mere granular detritus, or—
5. Fatty casts—if, on the other hand, the degeneration takes a fatty form; and certainly in the later stages of nephritis, when the acute inflammatory phenomena have subsided, fatty casts are more abundant. Lastly—
6. Large hyaline casts are formed by the coagulation of an exudation taking place into tubes denuded of their epithelium, and consequently with larger calibre.

Provided that the secretion of urine is fairly free, the relative abundance of any of these casts gives a rough estimate of the rapidity with which epithelium is dying and being shed. So that from a clinical point of view the abundance and the nature of the predominant form of cast may be able to give us some idea of the progress of the case. Thus blood casts will not be likely to predominate in any case that is improving; fatty casts rather point to a want of recuperative power in the kidneys, while a large total number of casts of epithelial origin point to a grave destruction of renal substance still going on.

Beyond these hints, casts are of comparatively little value in a case of acknowledged and definite nephritis; but it is in cases of less definite character, and more insidious onset, where albuminuria first draws serious attention to the renal function that the *presence* of casts is such an important point to determine. Thus, in the following conditions more especially (though in no case of albuminuria must repeated searches for casts be omitted), the presence of casts must give us serious concern for the safety of our patient.

1. Pregnancy.
2. Any abdominal tumour likely to cause pressure on kidneys or renal vessels.
3. Any case in which serious surgical procedures of any kind are contemplated.
4. Convalescence from, or presence of, scarlet fever, diphtheria, and in fact any acute illness, especially of microbic origin.
5. In a case hitherto regarded as functional albuminuria.

The presence of numerous casts shows that the albuminuria is probably due to something more than slight nutritive weakness of the kidney, and therefore *ceteris paribus* adds enormously to the gravity of the situation.

URATES

It is well to remember that this is the only deposit that occurs in urine (which has not decomposed) as the result of simple cooling, and hence when the means are not at hand for properly testing the secretion, if the urine was bright and clear on voidance, a patient's mind can often be set at rest at once by finding that the secretion was clear until it cooled.

We have already noticed that urates are often enough present in large quantities in health, under various conditions of exercise and weather, etc. Their pathological associations are as numerous as the whole nomenclature of diseases, for there is no illness which may not be associated with (? actually cause) a deposit of urates in the urine. As a matter of practical clinics, therefore, their diagnostic value or importance is not great, but we must generally suspect when urates are in excess

1. Febrile disorders of some kind, especially rheumatism ; or—
2. Alimentary disturbance, and especially if gout be in question.

In the former class they probably indicate an insufficiency of oxygen to burn up the increased waste products ; in the latter some slight disturbance of the course of the body's metabolism ; in either case they may be said generally to indicate the necessity for a free evacuation of the bowels, and due attention to the organs of digestion.

Their relationship to stone will be briefly referred to under that head.

OXALATES

These are comparatively unfrequent elements in a deposit, and but little is known of their causation beyond the fact that they may occur in quantity after a meal in which rhubarb or sorrel leaves has been freely partaken of. A few doses of nitro-hydrochloric acid will rapidly remove them from the urine, and the aching pain from the back, the latter being their only known symptom.

PHOSPHATES

Phosphorus in combination appears in the urine in four groups of bodies, viz.—

1. In combination with organic radicals in the shape of lecithin, protagon, etc., the existence of which is well recognised in the excretion; but the results of their estimation and the significance of their presence are not yet available for ordinary clinical work, and therefore nothing further will be said about them.
2. As the phosphates of the fixed alkalies Na and K.
3. As the phosphates of the alkaline earths Ca and Mg.
4. Triple phosphates.

On these last three groups a word or two may be said.

(2) *Phosphates of Sodium and Potassium.*—We have already noted these bodies as the chief cause of the acidity of the urine. The only other caution connected with them is to remember that they are so freely soluble in water that they *never* in untreated urine form any part of the very common phosphatic deposit. This caution is the more necessary, as a student almost invariably replies, "Sodium and potassium" if asked what phosphates are present in a deposit.

(3) *Phosphates of Ca. and Mg.*—These are the phosphates which are precipitated on boiling, or in a urine which is acid without artificial treatment. So long as they appear only on boiling, or as a scarcely appreciable quantity in an otherwise healthy urine, they may be neglected in diagnosis; but when they appear in distinctly measureable quantities they assume at once a position of greater importance, and a few points will be noticed (*vide* Phosphatic Diabetes).

(4) *Triple Phosphates.*—The name "triple," though sanctioned by long usage, seems to me somewhat badly chosen, because, unless

one is very careful to explain the nature of these crystals, students jump to the conclusion that triple means that three metals are combined with the phosphoric acid instead of understanding that all three atoms of hydrogen are replaced, two by the dyad Mg, and one by the monad Am, so that the formula is Mg Am PO_4 and aq.

The only clinical deduction their presence allows is, "This urine has decomposed."

Why this deduction?

Because there is no other available source for the Am except that provided by the decomposition of urea.

Then arises the all-important point, "Did this decomposition take place inside or outside the body?" If outside, and only after some time, it represents merely a normal event in the natural history of an organic substance exposed to the action of micro-organisms. If inside, or within say an hour of being passed, we know at once that either the bladder or renal pelvis (or possibly both) is in a very unhealthy condition, which, if not speedily improved, is likely to lead to most disastrous consequences.

Very slight attention to the history will usually suffice to clear up the point, especially if the question be asked, "Are you sure the urine was quite clear on being passed, and only clouded after standing some time?" Earthy phosphates without ammonia, pus, and chyle, are the only fallacies in that which is passed cloudy, while urates will be the only fallacy in that passed clear, and each of these can very readily be differentiated (*vide* Chemical and Microscopical Tests).

We have already (p. 200) discussed the diagnosis of kidney or bladder as the seat of the trouble; and for relief of the symptom (not necessarily of the disease) local injections of hot boracic lotion and benzoate of ammonia in 15 grain doses are, in my experience, the most likely means.

URIC ACID

On what may be called the pathology of the uric acid diathesis of gout and of gravel and nephrolithiasis, so much has been written, so many theories have been propounded, and in their turn confuted, that it seems desirable to state what is accepted as fact, and separate it from the more theoretical and disputed points. This we may do in parallel columns, so as to draw more pointed attention to the arguments.

Points agreed to by all, and accepted now as facts.	Points about which conflicting theories and statements are still being made.
As to Uric Acid itself	
1. That uric acid is one of the possible products of the decomposition of the complex nitrogenous bodies which exist as component parts of the various tissues of the body, or which are taken into the body as food stuffs.	Whether the food (*i.e.* that part of it which has not entered, or will not enter into the molecular constitution of the body tissues) or the tissues is the ultimate source of the uric acid found in the urine of man.
2. That it is practically the only representative of nitrogenous waste products in some animals, *e.g.* birds and snakes.	Whether the urates of snakes' urine, etc., are identical in nature, as well as composition, with those of human urine.
3. That it is present in certain small amounts in the urine of every healthy human being.	Whether it should bear any constant proportion to the urea, and how to account for the varying amounts and proportions found.
4. That its chemical composition is $C_5H_4N_4O_3$, and thus contains a less proportion of O than urea CH_4N_2O.	Its exact chemical relationship to urea, *i.e.* whether in the human body urea can be, and is made from uric acid, or *vice versa*; or whether they appear side by side, as it were, as independent results of the same chemical processes of decomposition; or whether each requires a separate series of reactions for its production.
As to Locality of Manufacture	
5. That it is manufactured somewhere in the body.	Whether in the liver, the tissues generally, the blood, the spleen, or the kidney.
6. That it exists in the blood of a patient who has gout.	Whether it exists in the blood of a healthy individual.
As to its Clinical Relationships	
7. That there are two well-marked clinical types of trouble asso-	Whether these two diseases must be reckoned as primary renal affec-

Points agreed to by all, and accepted now as facts.	Points about which conflicting theories and statements are still being made.
ciated with uric acid, and that these two, viz. gout and gravel, may and do occur separately.	tions, or whether the kidney suffers only secondarily.
8. That heredity plays a strong hand in the production of gout or gravel, but frequently alternations of the two in successive generations are met with.	Whether heredity or personal environment (in its widest sense) is the stronger factor.
9. That cartilage and fibrous tissue do become infiltrated with crystals of urate of sodium.	Whether this deposition of urate causes the painful affection of a given joint; Whether such deposits can be reabsorbed; Whether so-called acute gouty arthritis is not really acute neuritis; Whether the deposition is a cause or consequence of local necrosis.
10. That per pound of body weight children excrete more uric acid than adults.	Whether this indicates freer excretion only, or more active formation; in fact, its meaning.
11. That gout is as rare in children, compared with adults, as stone and gravel are common.	The essential meaning of this.
12. That gout, lead poisoning, and cirrhotic kidney occur in the relationship of predisponent, excitant, and resultant in a far larger proportion than mere coincidence will account for.	Which of the three is predisponent, excitant, or resultant; and whether each one may not act in any capacity at times.
13. That diet can influence gout and gravel.	Precisely what kind of diet is best for gouty patients.

In conclusion, we may say that until some of these points are authoritatively settled by a unanimous agreement, above all, the tissue source, and seat of manufactory of uric acid, the only practical clinical points that may be safely deduced are:—

1. That uric acid, by its presence in the body in excess, is

capable of working mischief, or at least that uric acid is the outward and visible sign of a vital metabolism which is incompatible with perfect health.

2. That if we find it in excess in the urine or body, we should recognise its potency for evil, and try by some means to check its production in, or ingestion into, the body.

3. That as regards diet, exercise, and even drugs, each case must be treated at first more or less experimentally, and later by the light of such knowledge as experience of the individual case may give. Sodium salicylate and iodide, with moderation in butcher's meat and alcohol, are the most promising measures with which to commence to treat an unknown patient.

GLYCOSURIA

With regard to sugar in the urine, we are in a very similar position to that in which we stand in the case of uric acid. Many facts require explanation, and many theories have been brought forward in consequence. Were it not for the resolute opposition of Dr. Pavy, who has made diabetes the special object of a life-long study, we might say that there is complete unanimity in the views of the morbid physiology of glycosuria. A similar arrangement to that we adopted in the case of uric acid will again serve to emphasise fact and theory.

Fact.	Theory or Doubt.
1. That sugar does occur in pathological quantities in the urine.	Whether minute traces of sugar are or are not normal in the urine.
2. That pathological condition may be— (*a*) Temporary, as after chloroform and experiment. (*b*) Intermittent, as in some elderly patients. (*c*) Permanent till death, as in many cases of diabetes in both young and old.	Whether glycosuria and diabetes can be separated as two distinct conditions ; and supposing they can, what is the real and precise relationship between the glycosuria of diabetes and the remaining characteristic symptoms of that disease.
3. That in many cases, especially of Groups (*a*) and (*b*), it is present without the usual associated symptoms of actual diabetes ever occurring ; but	

Fact.	Theory or Doubt.
in many commencing as apparently simple glycosuria the more severe symptoms do eventually occur.	
4. That diet, especially with regard to carbohydrate material, does influence very markedly almost every case of glycosuria.	What is the precise connection between food and sugar in the urine.
5. That the liver contains glycogen during life; that this glycogen is derived mainly from carbohydrate food material; that after death, if no precautions are taken, the liver can and will convert nearly all the glycogen into sugar.	It is on this point that Dr. Pavy is especially insistent, viz. that we have no proof that what the liver does after death it is capable of doing during life; and hence he denies the more commonly accepted view, viz. that the liver seizes upon all carbohydrate material, converts it into glycogen, and then reconverts it into sugar, according to the needs of the body.
6. That in many cases the liver is in some way the main seat of those disturbances in metabolism which result in glycosuria and diabetes.	The real nature of the disturbance, whether it is— (*a*) Merely excessive escape due to excessive circulation through the blood vessels. (*b*) Excessive escape due to excessive action of the cells of the liver. (*c*) Faulty metabolism of the cells, so that a wrong sort of sugar is produced, useless for the tissues. (*d*) Faulty metabolism in not seizing the sugar brought to the liver and converting it into glycogen.

As regards Morbid Anatomy

7. That in some cases of diabetes the pancreas is found diseased.	Whether the disease of pancreas means a fifth view of the morbid physiology of diabetes, equivalent to a diminished destruction of

Fact.	Theory or Doubt.
	sugar by the internal secretion of the pancreas.
8. That in the great majority of cases of diabetes no naked-eye appearances are found capable of explaining the causation of the trouble.	Whether there are not such microscopical changes in the medulla as might explain altered metabolic power on the part of the liver
9. That the coma from which the great majority of diabetics die receives no adequate post-mortem explanation.	The precise nature of the substance which, by its presence in the blood, causes the coma.

In conclusion, whatever views may be held on the disputed points in the pathology of diabetes, the diagnostic points are very simple, for the facts are so strong that no objection can be raised to the statement, "If sugar be constantly present in the urine—for a longer period than, say, twenty-four hours—in sufficient quantity to be detected by thirty seconds' boiling with Fehling's solution, there is a serious pathological condition present which requires the careful attention of the medical man." (For one or two further remarks of a clinical character, *vide* Diabetes.)

URÆMIA

This term was originally invented to express a series of symptoms due to a supposed excess of urea in the blood; we now use it in a wider sense, or rather in a different sense, to cover the symptoms produced by the retention within the blood (or tissues) of waste products in general, which should be eliminated with the urine. Thus far everyone is agreed, but when the further question is put—"What is, or are, the particular waste products?"—no answer is forthcoming except negative ones. It is not urea, nor uric acid, nor potassium [1] (*vide* footnote), nor water, nor inorganic salts, nor kreatin, in fact, it is not any single ingredient of the urine which has hitherto been isolated.[2]

The nearest probability would be a suggestion that each organ and tissue is responsible for its own share in the production of the total phenomenon; the special waste products of each (whatever

[1] The latest view of all is that uræmia is due to retention of potassium salts.

[2] All these points are conclusively proved by M. Bouchard's work on auto-intoxication, translated by Oliver.

their nature) acting as a hindrance to the proper functioning of itself (and probably to a varying extent of other organs).

Diagnosis of Uræmia

To associate its occurrence into the following groups will, I think, best conduce to its diagnosis.

Group A.—Acute and severe, probably, though not certainly, producing a rapidly fatal termination to—

(1) A known and watched case of kidney disease.

(2) A known and watched case of other disease in which urinary symptoms are now, or have been all along, prominent, *e.g.* cardiac disease with suppression.

(3) An unknown and unwatched case of sudden illness of convulsive or comatose type in a patient previously supposed to be in good health.

Group B.—So-called chronic uræmia, in which the symptoms, though possibly acute enough and severe enough in themselves, pass off or yield to appropriate treatment, and recur again and again over months or, it may be, years.

Of Group A, Nos. (1) and (2), I have nothing to say beyond what will be found in every text-book. The symptoms are fairly uniform, starting with a violent headache or sudden blindness, or acute vomiting, or diarrhœa, and rapidly passing into convulsions, coma, and death ; or it may be sudden convulsions or coma, passing into death. In any case, the only difficulty in diagnosis rests in bearing in mind the great possibility, or rather probability, of uræmia supervening in a certain class of diseases.

Group A, No. (3), presents great difficulty in diagnosis, but as the condition is one presenting great likeness to alcoholic and other poisoning and cerebral disease, it is fully discussed under the heading with which the lay press has made us unhappily familiar, viz. "Drunk or Dying" (*q.v.* Chap. IX.).

Group B is the group of chronic uræmias to which I wish to draw special attention, as I feel convinced that many cases of it pass unnoticed or called by another name, for the simple reason that students are so accustomed to the idea of the acute, rapidly-fatal symptoms as the only form of uræmia, that they overlook the "fringes" (to borrow Dr. Goodhart's phrase) of the condition.

The late Sir Andrew Clark, I believe it was, who coined the expression "renal inadequacy," and this is the condition which

underlies and explains many or all of the following symptoms so commonly found in elderly, and even young people, whose urine is habitually only just within the physiological limits of the product quantity × specific gravity. I refer to attacks of indigestion or diarrhœa without obvious dietetic irregularities, to attacks of deafness, giddiness, etc., swimming in the head, so often thought of as precursors of apoplexy; to severe headaches of a prostrating character, to transitory paræsthesiæ, felt in parts of the body or limbs, or pareses of the same parts; to a frequent feeling of malaise and general despondency; nay, I will go farther and say, without fear of contradiction, that this same renal inadequacy is at the bottom of the known experience that operations on kidney subjects do badly, that it explains the liability of many patients to chronic bronchitis and emphysema, pneumonia, pleurisy, pericarditis, peritonitis (whether serous or suppurative), cardiac failure; that, in fact, it is the real meaning of those so-called intercurrent affections, not excluding acute nephritis, which so frequently close the scene in cirrhotic kidney. It is the circulation of imperfectly depurated blood that renders all these organs so liable to break down with acute inflammatory mischief. This persistent and steady, though it may be very slow, accumulation of waste material is as essentially chronic uræmia as is the rapid accumulation acute uræmia.

As far then as diagnosis is concerned, the rule may be emphatically laid down that in all diseases of the above character the urine should be systematically examined every day, and especially is this necessary if the patient is over forty. An elaborate analysis is not required, but the total quantity and specific gravity are essential for a correct appreciation of the output of waste organic material.

Suppression *v.* Retention

If there is reliable information that very little or no urine has been passed, say for twenty-four hours, these two conditions cannot easily be confused. An abdominal examination must be made: this will or will not reveal the presence of the distended bladder; in either case, the catheter must then be passed to relieve the symptom if the bladder be distended, or to prove the absence of urine. The only exceptions to the whole of the above rule and treatment are occasional cases of pure hysteria, in which the pleasures of instrumentation overcome the discomforts of a full bladder. Such cases require great tact in handling, but offer no diagnostic difficulties.

Difficulties in diagnosing retention may occur when overflow is

also present, or when micturition is alleged to be natural. The former occurs commonly enough in puerperal cases (I have known a distended bladder under these circumstances called acute metritis), and should also always be held in mind when diseases of cord and brain are being treated. It will, of course, be usually associated with local pain, directing attention to the bladder, but if unnatural anæsthesia be present, a distended bladder requires to be borne in mind and looked for. The latter condition, *i.e.* distension, with alleged natural micturition, is practically confined to elderly male patients with prostatic trouble. It is apt to be very misleading, owing to the gradual acquirement of a condition of tolerance for increasing quantities of residual urine. I have seen a patient with bladder distended to the umbilicus who assured me that he could not pass me any water because he had micturated just before his visit. He was totally unconscious of his urinary condition, which proved fatal within a few weeks.

The causes of suppression of urine, *i.e.* of anuria with empty bladder, may thus be tabulated :—

A. Mechanical blocking of ureter, due to	Stone (*vide* Stone in Kidney). Carcinoma,—previous hæmorrhage, and tumour. Tubercle,—previous pus, and hæmorrhage. Blood clot,—previous hæmorrhage.
B. So-called functional from kidney conditions, due to	Heart failure, or other form of venous back pressure,—cardiac bruits, with obvious back pressure, or swelling and tumour of abdomen. Active irritation, such as that caused by drugs,—history of taking a drug. Actual inflammation,—other signs of acute nephritis, anæmia, puffiness of eyelids, etc.

The causes of retention, *i.e.* anuria, with a full bladder, are again :—

A. Mechanical obstruction.	Position diagnosed by the catheter; uterine condition also to be thought of.
B. Functional incapacity of expulsion.	Cause to be sought in the nervous system, or in previous history of over-distension.

BRIGHT'S DISEASE

Before saying anything as to the diagnosis of the various forms of Bright's disease, it would be well to define the term. There are

then four fairly well-marked types of it from a clinical point of view:—

1. *Acute Nephritis.*—The word "acute" as used in medicine sadly needs accurate definition: sometimes it has mainly reference to time, *i.e.* with well-marked definite onset, without reference to the severity of the symptoms; sometimes it is used to denote more the intensity of the symptoms, *i.e.* in our present illustration the smoky or bloody urine. Acute nephritis is practically always due to a toxæmia, *i.e.* to the efforts of the kidney to secrete from the blood some substance which actively irritates the kidney cells in the process of separation, *e.g.* the poison of scarlet fever, diphtheria, or substances produced in the blood by a condition popularly known as a chill.
2. *Consecutive Nephritis.*—This is a convenient term to designate the condition of a patient who has (*a*) at some previous period suffered from acute nephritis, and has never since been quite free from urinary changes; (*b*) has drifted into a condition of renal disease without marked onset, as in some of the scarlet fever cases not under careful observation, and (?) some cases of earlier albuminuria simplex (*q.v.*); or (*c*) has his kidneys irritated by local conditions, such as stone, simple blocking or kinking of the ureter, and all forms of surgical kidney. The group is obviously a mixed one, and most of the cases will in practice naturally have a specific name applied to them, but they all have one point in common, that if they last long enough, and if the essential kidney structure does not wholly disappear, they lead eventually to the form of granular kidney known as the small white or secondarily contracted kidney.
3. *The Cirrhotic Kidney*, of which one type is the later stages of Group 2, and the other is the small, red, granular kidney so frequently associated with degenerate vessels and large heart, with age over forty, with gout, and with lead poisoning. It must be ever constantly borne in mind that either type may at any moment blaze up into an acute nephritis; in fact, this is the commonest event in their course.
4. *Large White Kidney.*—A disease just as much *sui generis* and absolutely independent of other kidney trouble, as is, say, typhoid fever or cerebral glioma. It has a rapid history, with an invariably fatal termination within three to six

months. It never starts from, or turns into anything else, and may be called acute degeneration (fatty changes preponderating) of the kidneys.

As to their clinical course and main danger signals, these four types may be briefly epitomised as follows:—

Acute Nephritis threatens life chiefly in its early days, and that almost entirely by suppression and acute uræmia. If this stage be passed, we may then say that the lapse of nine to twelve months will show whether the case will end in complete recovery or will drift into one of consecutive nephritis.

Consecutive Nephritis.—Of the sub-groups (*a*) and (*b*) the duration is commonly one of years, though of course often much less, in which the patient shows important and marked changes in the urine (albumen and casts). The main clinical features of the condition will be occasional puffiness of the face, especially in the lower eyelid, general flabbiness and malaise, and, above all, anæmia, to which I would draw special attention as the main element in the disease. So long as the patient keeps a fair colour, or the anæmia does not advance, so long are there good hopes of prolonging life; but with the advance of blood impoverishment must come increased anxiety as to the advent of severe uræmic symptoms, of which the anæmia is the advance guard and warning beacon. Of the sub-group (*c*) the dangers are much the same, for they essentially depend upon disorganisation of the kidney, but the prognosis has two elements in it—first, "How far has the destruction of the kidney substance progressed before surgical measures are adopted?" and, secondly, "Is the primary cause of the trouble one which can be permanently cured by these surgical measures if they are applied?"

Cirrhotic Kidney.—The first group is sufficiently sketched under Consecutive Nephritis, of which, indeed, it forms merely the concluding chapters. The other type of cirrhotic kidney, *i.e.* the independent form without previous history of urinary trouble, can scarcely be said to have a clinical course. It begins so insidiously that it hardly comes under notice until the thickened arteries and the hypertrophied (probably also dilating, *vide* Morbus Cordis) heart are discovered on the routine examination of a patient who comes before us struck

down by apoplexy, or affected with one of the diseases already mentioned as essentially uræmic (*q.v.*) in nature. The last scene is always either of this nature, or else a simple uncomplicated uræmia, which in turn is brought about either by an absolutely too great organic destruction of kidney tissue, or by an acute nephritis, which renders a still organically adequate kidney functionally inadequate.

Large White Kidney.—This has for years been described as a form of chronic Bright's disease, and included amongst those cases for which I have adopted the term Consecutive Nephritis. A comparison of the accounts of writers thus describing it, combined with my own post-mortem experience, have compelled me to claim for it a course which is, at any rate, acute enough when measured by time, and also by urinary symptoms. If at the post-mortem the kidneys (not being amyloid) are very white, large (say together over 14 ozs.), and especially if the capsule strips easily without tearing the substance at all, but leaving a perfectly smooth surface to the organ, then the clinical history shows only a four to six months' duration of illness at the outside, and frequently one of only six to ten weeks. The main features of the illness are always the same, viz. very marked anæmia, excessive œdema, especially of thighs and legs (large white legs, large white kidney, is a pathological proverb), and a urine such as is tabulated below; and these serve to distinguish this affection during life from any other except the absolutely final chapter of a consecutive nephritis which, if diagnosis is required, can be readily recognised by its commencement. The invariable ending of large white kidney is acute uræmia.

This brief sketch shows plainly enough that all forms of Bright's disease have a distinct tendency to drift into serious uræmia, the only exception being some cases in Group (*c*) of Consecutive or Secondary Nephritis, provided that the local cause of the nephritis is one which is capable of complete removal by surgical aid, and also provided that the condition is ascertained and removed before extensive, serious, and progressive destruction of renal tissue has been set up. Our first object, then, when a patient presents himself with a condition of urine indicating damage to the kidney, is to ascertain whether such a removable cause is present or not. This will, however, be more conveniently discussed in the next section;

in the present one we will discuss the diagnosis of the uncomplicated forms of Bright's disease.

A slight inquiry into the previous health and urinary history of the patient, combined with a knowledge of the onset of the present illness, will speedily separate—

Primary acute nephritis Secondary acute nephritis (as an incident in chronic) Large white kidney	on the one hand from	Simple chronic nephritis of either type without the acute exacerbation.

They will also separate very distinctly the primary from the secondary acute nephritis. The profound anæmia of very rapid onset, combined with the extreme œdema, are usually striking enough to separate large white kidney from either form of acute nephritis. Should these general indications not be sufficient, the condition of the urine tabulated below will settle the matter. In the more chronic conditions a keen eye to note progressive asthenia and anæmia, and a careful analysis of the urine from day to day for a short period, are the important requisites for estimating the stage which the sufferer has reached on his road to uræmia.

URINE IN BRIGHT'S DISEASE (AND IN LARDACEOUS KIDNEY)

Acute.	Consecutive and Cirrhotic.[1]	Large White.	Lardaceous.
Quantity—			
Much diminished, sometimes to suppression.	Increasing in proportion to the change towards cirrhosis; diminishing at the last.	Much diminished, but not to suppression.	Usually increased, but not always.
Blood—			
Invariably present in visible amount; smoky or bloody.	Absent without acute exacerbation.	Only detected by the microscope.	Absent.
Sp. Gravity—			
Increased.	Diminished, even if quantity diminished.	Probably below average.	About normal.

[1] These changes are independent of pus, blood, crystals, etc., produced by any possible local trouble, and refer to the nephritis only.

Acute.	Consecutive and Cirrhotic.	Large White.	Lardaceous.
Albumen—			
Much, often nearly solid, probably coloured by blood.	Variable, but generally not more than a trace unless serious condition coming on, often quite absent.	From half to solid, always much, not coloured by blood.	Variable, but always considerable, *i.e.* one-fifth or more.
Casts—			
Blood, epithelial, etc. (*vide* under Casts).	Very few, hyaline or fatty, more numerous if acute exacerbation.	Plentiful, some epithelial, but mostly fatty and hyaline.	Sometimes none, may be many, and are said sometimes to show lardaceous changes.
Colour—			
Dark, concentrated, smoky, or red.	Very light, except in acute exacerbation.	Natural.	Usually lighter than natural.

In the daily analysis the points of especial importance are the total quantity and the specific gravity, the relative abundance of casts and other debris. The former giving us the product of specific gravity by quantity, which is a rough indication of the output of waste material; the latter giving a fair estimate of the activity of renal destruction. In comparison with these two indications the amount of albumen sinks into insignificance.

We may now discuss those conditions of the urinary tract which are liable to cause by their continuance a consecutive or secondary nephritis, or to be mistaken for Bright's disease owing to certain urinary changes produced by them. They may be enumerated as follows:—

Group 1. Preputial or urethral stricture, prostatic enlargement or other affection of the prostate, stone in the bladder, etc., in fact, peripheral obstacles to the escape of urine from the bladder, as well as certain ulcers and growths of the bladder itself.

Group 2. Similar affections of the ureter arising in its structure, or affecting it from without.

Group 3. Affections of the pelvis or substance of the kidney—

Stone.	Hydronephrosis from unknown causes.
Tubercle.	
Malignant growths.	Hydatids and other rarer affections.
Undue mobility.	

The differential diagnosis of the members of Group 1 is essentially surgical, and does not ordinarily present much difficulty, as the prepuce, penis, urethra, prostate, and bladder are open to direct examination and investigation by the eye, finger, bougie, and cystoscope. From the urgency of their symptoms they compel surgical interference, whatever be the functional capacity of the kidneys as a factor in prognosis, so that the possibly associated Bright's disease may be temporarily left out of the question.

The presence of a member of Group 2 can only be surmised by the positive fact of the presence of pathological urine, or a tumour in the abdomen, combined with the negative fact of the absence of disease from the bladder down to the prepuce, leaving us to infer that the trouble is situated in ureter or kidney. The only cases in which, then, the ureter would be suspected would be the disappearance of a known collection of pus with profuse pyuria appearing, or the discovery of a hydronephrotic tumour. In practice, however, it is impossible to distinguish even these cases from trouble at the hilum of the kidney.

The third group, viz. affection of the pelvis or substance of the kidney itself, requires more detailed examination; and we may commence with the three more ordinary affections, viz. stone, tubercle, and malignant disease. Though not absolutely germane to the subject of differential diagnosis, there is one clinical fact that, on account of its tremendous importance, cannot be omitted, viz. that any one of these three may—and not as a mere pathological curiosity, but even with some little frequency—be present without causing either a pathological condition of the urine, or a tumour in the abdomen; not only so, but the patient may even die of obscure symptoms, probably uræmic in nature, without the kidney being suspected, until a post-mortem examination reveals the most extensive destruction of one organ, or even of both of them.

We are, however, now assuming that some or all of the following local indications are present—blood, pus, albumen, casts, and other deposits in the urine, pain, either colicky or persistent, tumour in the abdomen; and we have to consider, from their various combinations, which is the most likely lesion present:—

The following table shows the chief diagnostic indications:—

	Stone.	Tubercle.	Carcinoma.
Quantity of urine.	During colic often suppressed, otherwise unaltered or increased.	Usually unaltered, if any effect it is diminished.	Also usually unaltered.
Blood.	Moderately frequent, but usually not severe, and quite intermittent.	Often a smart hæmorrhage, less frequently repeated than in other conditions.	Hæmorrhage not infrequent, but more likely to be a continual oozing, therefore blood pretty constantly present.
Pus.	Not common in any quantity, unless urine is alkaline and ammoniacal.	Often very profuse indeed, and that with quite acid urine, and without necessarily severe symptoms.	Not profuse as a rule; if present in quantity, severe features are usually present.
Albumen.	Unless associated with either blood or pus is really an indication of Bright's, and, as such, has much importance.	Practically only in proportion to pus.	In proportion to pus and blood, and possibly some nephritis, but obviously of no real import.
Other deposit.	Uric acid, if any, and minute stones.	Tubercle bacilli in the pus.	Fragments of growth possibly, but they are rare.
Pain.	Often none; sometimes a constant dull ache, but renal colic the typical form; active jolting almost	Dull ache most usually, if any at all, but ordinary attacks of colic not unknown.	Dull ache or acute local pain; typical colic very rare.

	Stone.	Tubercle.	Carcinoma.
	invariably causes exacerbation.		
Tumour.	Of stone *per se* never present, but may be a hydronephrosis.	Kidney often large, may be felt as a tumour; kidney frequently hydronephrotic.	Tumour if felt more irregular, much less like a kidney in shape; may bulge into loin.

This table will well bear a few clinical remarks. The most important practical general statement, and the one most humiliating to medical science, is, that after all points have been fully considered, the diagnosis will sometimes still remain in doubt; gravel, bacilli, and fragments of growth, the three almost pathognomonic signs, may each and all fail us, and we have to call to our aid exploratory surgery for the relief of symptoms serious in themselves, but of unknown causation.

Apart from these doubtful cases of impossible separation, we may say a few words on the individual conditions.

Stone in the Kidney

It has already been mentioned that the kidney may be entirely disorganised by stone, without its presence being suspected from any active phenomena; but we are now dealing with an analysis of actual symptoms, amongst which renal colic (intermittent in type, made worse by jolting movements, passing to the testicle) and intermittent hæmaturia (almost constantly associated with colic) are the two most suggestive. It is worth bearing in mind (in the presence of colic with suppression of urine) that in a *first* attack of colic from renal calculus both ureters are never blocked at the same time, and we therefore have strong hope under such circumstances of a first attack that the flow of urine will be re-established within a short time, even if the stone be not passed; but if the patient be suffering from an attack of renal colic, and there be a history of similar previous trouble, especially if of great severity, then the probability of both ureters being blocked is considerably increased, and our anxiety for early mechanical relief proportionately acute. This is especially the case when we remember that uræmia from

obstructed ureters is very insidious — a little increased general weakness in the patient, an occasional twitch in one or two muscles, no obvious ingravescence of these till quite suddenly drowsiness and fatal coma set in.

Tuberculosis of Kidney

This is most commonly a secondary affection, or rather it would be more accurate to say the kidney—if tubercular—is rarely the only tubercular focus in the body.

1. It may be part of a miliary attack, in which case it will only be recognised on post-mortem examination; it possesses no clinical diagnostic interest.

2. It may be associated with obvious tubercle of the lung. Here it will be recognised, if at all, by aching pain in the loins and urinary alterations — albuminuria, hæmaturia, or pyuria (*q.v.*). In such a case its recognition is more important from a prognostic than from any other point of view, for advanced phthisis is a strong contra-indication to any serious operative interference, and I have never seen cured, as opposed to merely quiescent, tubercle of the kidney.

3. Even when confined to the genito-urinary tract, the kidney may be only secondarily implicated by extension from the bladder. Here, frequent micturition, pain in penis, and pyuria will have attracted attention, and the cystoscope will have assured us that the bladder at any rate is implicated; renal casts and cells found by the microscope, and a continuance of pyuria after the bladder trouble has healed, will be the chief indications that the kidney itself is also implicated. Lastly, should the case be one of genuine primary tuberculosis of the kidney, the diagnosis will be formed somewhat as follows: an aching or even an acute pain in the loin will have led the patient to seek for advice; inquiry will then be specially made into the urinary function; or, failing any special complaint, the routine examination of the urine will have led us in the same direction, viz. to suspect the kidney. Inquiry and examination will then have revealed some features of great probability in diagnosis —pain, tenderness, hæmaturia, pyuria, etc. Lastly, to complete the diagnosis, bacteriological examination of the urine, or its deposit, will frequently enough be necessary, and in suspicious cases cannot be omitted.

N.B.—One ureter may be completely blocked by causation, so that the urine may be natural in quality, and possibly also in

quantity; hence we must not too hastily acquit the kidney if the previous history or local indications point strongly in that direction.

Malignant Disease of Kidney

Like neoplasms elsewhere, may be primary or secondary. If the latter, diagnosis is very much a matter of indifference in clinical work; the points are identical with those of a primary case, clarified considerably by the presence of the original growth. Primary malignant disease—sarcomata are tolerably common in children—can only be recognised by a process of exclusion; the hæmaturia, which is far and away the commonest symptom, being common to at least three renal affections, whose differential diagnosis has been considered above. Unless, then, we can find a tumour in the loin or abdomen, or at least some gross irregularity in the outline of the kidney, stone and tubercle must be first excluded. The cystoscope may show that the blood comes from one ureter only—a suspicious circumstance in doubtful cases, for it almost certainly excludes blood conditions, and thus renders local disease of some description almost certain.

PHOSPHATIC DIABETES

Is too rare a disease, at any rate in well-marked form, to require any prolonged discussion here, but I mention it to emphasise the one essential point in its diagnosis, viz. that the phosphates must appear in the urine without any treatment of the fluid—heat, alkalies, etc.—and in the absence of decomposition. In a typical case they will form a deposit equal to one-quarter, or even one-half the bulk of the fluid. The patient will have few or no complaints to make except of general flabbiness, langour, and want of energy. Routine examination of the urine will complete the diagnosis so far as we can at present go.

DIABETES INSIPIDUS

Precisely the same remarks apply to this trouble. It is a rare disease, of practically unknown causation, and needs only mention to emphasise the caution not to call mere temporary polyuria by the ominous—to the laity—name of diabetes. The essential point

is that, without rhyme or reason—occasionally a blow or shock causes it—an otherwise healthy person shall somewhat suddenly begin *and continue* to pass enormous quantities, from 200 ozs. upwards, of very light urine with a very low specific gravity—1002-1005—and no abnormal constituents. This, and a corresponding thirst, are the only real diagnostic elements.

CHAPTER VII

AFFECTIONS OF JOINTS

The symptoms and physical signs of a joint trouble are, with one, or possibly two exceptions, viz. hysterical knee and the pain in the knee when the hip is the seat of disease, sufficiently obtrusive to leave no room for diagnostic difficulty in deciding which joint is affected; but the precise structures attacked, and the extent to which they are likely to be disorganised or destroyed, with the consequent future utility of the articulation, will commonly, and speaking in general terms, largely depend upon the third step in diagnosis, viz. the exact etiological factor at work. Treatment, too, will very much depend upon the same factor for its nature and degree of activity. Compare the almost passive expectancy of the local *treatment of the joints* in simple rheumatic fever with the urgent necessity for evacuating a joint full of pus. Thus, both for prognosis and for treatment, exact diagnosis is of immense importance.

The affections, the diagnosis of which I propose briefly to discuss, are:—

Charcot's osteoarthropathy of tabes dorsalis.	Rheumatic gout.
Flat foot.	Rheumatoid arthritis.
Gout.	Synovitis acute.
Gonorrhœal rheumatism.	,, chronic.
Hysterical joint troubles.	Tubercle of joints.
Rheumatism.	Traumatism.

Some of them require only to be borne in mind for diagnosis to be at once evident, while in other cases a precise determination of the factors at work will remain in doubt after all tests have been

applied. We will commence with an analysis of the symptoms and physical signs as the simplest introduction to exact diagnosis.

A patient who has, or thinks he has, disease in or of a joint will come before us complaining of one or more of the following symptoms: pain (spontaneous, or on attempted movement), stiffness, grating, swelling, heat, or redness.

Pain.—This is a feature common to all cases, with one exception, viz. Charcot's joint, which is almost invariably (but not quite without exceptions) a painless trouble. The curiously misplaced reference of pain to the knee (through the obturator nerve), when the hip is the seat of serious trouble, must not be overlooked; the apparent shortening—tilt of pelvis—and physical examination of hip will usually clear up difficulties when nothing is found in the knee. For the rest the character and severity of the pain give us a strong guide to the acuteness or chronicity of the joint trouble. Thus, in acute gout, or a pyæmic joint, the pain is sudden in onset and agonising in its character. In rheumatism it is usually not so intense, but more wearying—a constant dull ache. When the bones are affected the pain is much worse at night, with jumping and starting of the limb on falling asleep. In more chronic affections, such as chronic synovitis, rheumatoid arthritis (in its chronic forms), pain is comparatively slight except on use, and other features will be of more use in diagnosis. The *exact* situation of the pain is a matter of great importance, for an abscess in a bone near a joint (especially true of the knee) has frequently been mistaken for joint trouble. If care be taken to examine for local tenderness, especially on tapping the *bone a little way off the joint*, the error will be likely to be avoided.

Stiffness.—May be of any degree, from the absolute rigidity of bony anchylosis to a little difficulty in commencing movement. In its milder forms, associated with some pain and aching, it may be merely the stiffness of some unwonted exercise. When this is the case it will usually be found that it is really the muscles and not the joint (though the latter has been accused by the patient) that is the seat of the pain. In any case, there will be the history of the exercise to guide us, and in a few hours, or days at the outside, it will subside. On the other hand, it may persist or get worse, and then we are faced with two alternatives: (1) it may be that the exercise (if there be a history of such) has excited a slight attack of synovitis: there will then be detectable a little effusion in the joint, or local heat, or grating near the joint (teno-synovitis), with considerable pain; (2) especially if there has been no unusual exercise

we must be on our guard, and look out for an oncoming attack of sub-acute or acute rheumatic fever. The thermometer will in this case show a slight (may be severe) pyrexia up to 100° or 101°. If there is no rise of temperature, the nervous system must be carefully examined, especially for sensory changes and, round the joint, atrophy of structures, etc. Should we still get a negative reply to our investigations, we have probably to deal with a subject of the arthritic diathesis (*vide* p. 244).

In its more chronic and definite forms the stiffness only appears *after an attack of actual joint trouble*, and then becomes chiefly of surgical interest, to ascertain what precise structures are involved, and how the stiffness can best be relieved. By this time, *i.e.* with a chronic stiffness, the history will probably have cleared up the etiology; but if this be unobtainable or unreliable, we may bear in mind that (*a*) bony anchylosis is most likely to have arisen from severe traumatism, recovered tubercle, or pyæmia, doubtfully including gonorrhœa; (*b*) bony obstruction without union, from rheumatoid arthritis, or from a Charcot's joint with excessive production of bone; (*c*) thickening and stiffening of ligaments, etc., from gonorrhœal rheumatism, or chronic rheumatism, and possibly gout; (*d*) stiffening of skin and other subcutaneous structures, from traumatism (burns, etc.), in all of which characteristic features are nearly sure to be present (*vide* below under the appropriate heading). To determine whether bony union as opposed to bony obstruction is present complete general anæsthesia is often essential.

Grating.—May be due to loose bodies in the joint and to acute synovitis, but in these two affections it can be produced by such manipulation of the joint as does not involve movement of the articulation. In the former case the history of the attack from which relief is sought is very characteristic. Possibly grating may have been previously perceived, but then has followed sudden fixation with intense pain. In both of them the sensation conveyed to the hand is very different—less harsh, more like a tremor—to that which is felt when the cartilage is eroded, and two bony or rough uratic surfaces are rubbed on one another. This latter form of grating is only felt in gout, rheumatoid arthritis, or Charcot's osteo-arthropathy, and then the remaining factors are of more importance (*vide* below).

Swelling.—The primary object in examining a swelling in or near a joint is to determine whether it is hard (bone, cartilage, or uratic deposit) or whether it is soft, probably fluctuating (synovitis, tubercle, sarcoma, aneurysm, etc.). Very slight examination will

soon determine this point. If the swelling be hard (due to bone cartilage or intraosseous sarcoma) it may be due to (1) Charcot's joint, in which case the lumps will be loose and movable on one another, and the joint will be usually more movable than natural; (2) osteoarthritis, when it will be more a lipping of the cartilage, an exaggeration of the natural outline of the articulatory ends of the bone, and the joint will have a less extensive range of movement than usual; (3) intraosseous sarcoma—this will be detected by the swelling being a little below or above the joint—of the bulk of the bone as opposed to its free edges—and possibly may give egg-shell crackling. A uratic deposit will also be hard, but its white appearance (or even discharge from a small opening) will scarcely allow of a mistake being made.

If the swelling be soft (or fluctuating) our first object is to determine whether it arises outside or inside the joint, or both. If purely outside a little care will usually reveal the outlines of the articulation, or a little pitting on pressure (the hip and shoulder offer almost insuperable difficulties in deciding this point), the articulation itself will usually work smoothly (in the hip and shoulder this will be the best test). If purely from within, or from both, the outlines of the articulation will be obscured more or less in some positions of the joint; this point is, however, more difficult in theory than in practice. In swelling outside the joint we may have to deal with aneurysm or sarcoma of the bone or bursal enlargements; the former, unless cured or ruptured, will not only be soft, but have an expansile pulsation of its own; sarcoma will have enlarged the bone some little way from the joint, while the contour of the joint itself may still be made out. Such are the principal points in typical cases, but in some unusual ones the diagnosis will require many examinations, and even then still be left in doubt; for Bursal Enlargements, *vide* below.

In a swelling which involves the interior and exterior of a joint, traumatism, gout, and gonorrhœal rheumatism come in chiefly for consideration. Traumatism will be determined at once by the history; for Gout and Gonorrhœal Rheumatism, *vide* below. When we have determined that we have to deal with soft material entirely inside a joint, pus, clear synovial effusion, and granulation tissue require to be differentiated. So far as the effusion itself is concerned we cannot decide between pus and clear fluid, but other points will leave little room for doubt between pyæmia and simple synovitis. Between an effusion and a growth of granulation tissue differentiation is usually tolerably easy, either by the pulpy semi-

fluctuating feel of the swelling, or (in the knee) by ascertaining whether the patella can be "rung" on the femur, *i.e.* whether we can feel bone knocking on bone, or whether some soft material comes in the way, as though the patella were depressed on to a soft cushion as it were.

Bursal Enlargements.—Are either (1) quite isolated, *i.e.* not communicating with the joint, or (2) provided with an opening into the synovial cavity. In the former case difficulties of primary diagnosis can hardly arise, for the swelling will be readily movable on the tissues which surround it and the joint; it will be incapable of compression, though probably fluctuating, while the outlines of the joint itself will be readily distinguishable, and its movements smooth. Communication with the joint cavity will be ascertained by examining the swelling in many positions of the articulation, when it will be found that in the position of greatest capacity—usually one of semi-flexion—the cystic swelling becomes more lax, its contents can be, partially at least, transferred to the joint and again expelled by a wide alteration in the position of the joint.

Heat—Distinctly appreciable by the hand as a contrast to the general heat of the skin, will only tell us that the condition is acute; in chronic joint troubles local heat is practically absent. Of acute affections, the heat is remarkable in gout and sometimes in pyæmia; in traumatism it is fairly well marked as a rule, but in rheumatism is either practically absent (milder or more chronic cases) or is very little noticeable owing to the great general heat of the skin.

Redness.—Is a pretty sure indication that the trouble is not purely intra-articular. Thus, it is scarcely, if at all, present in pure rheumatism (a synovitis) in simple synovitis, either acute or chronic; in gonorrhœal rheumatism (a pan- and periarthritis) it is commonly fairly well marked, but in gout it assumes its most intense and remarkable degree with a peculiar shiny condition of skin hardly seen in any other form of arthritis; in fact, if a bright red, shiny condition of the skin over and round a joint has appeared within a few hours, with intense agony, in a patient previously in fair health, it is almost pathognomonic of gout; the only alternatives are traumatism, of which the history will be obvious (and even here we must remember the frequency with which slight traumatism will excite acute arthritic gout), or less commonly pyæmia, and here there is not likely to be a freedom from previous symptoms of pyæmia, a definite source of the infection will usually too be fairly obvious, *e.g.* a wound, parturition, etc.

Individually, then, it will be seen that these local physical signs are rather frail reeds to rely upon for a diagnosis, but collectively (*vide* below) they form a strong foundation, and especially when considered in the light that the history of the case can throw upon them. In this history the following points are the most important :—

1. In the Family History.—Gout, rheumatic gout (probably rheumatoid arthritis), or rheumatism and its allies—chorea, morbus cordis, etc.—in very near relatives (*vide* below, Arthritic Diathesis).

2. In the Previous Personal History.—Excess of eating or drinking (especially beer) make gout seem more probable; chorea and definite cardiac disease make rheumatism likely; previous attacks of joint trouble similar or dissimilar to the present one will also have great weight.

3. In the History of Onset—

(*a*) Traumatism to the joint is obvious enough, but traumatism to a nerve (and other definite nerve diseases) must be noted as a possible causative factor.

(*b*) Exposure, either general or local (of a limb), is likely to produce a fairly sudden onset of rheumatism or acute synovitis.

(*c*) An attack commencing during sleep is *ipso facto* likely to be gout.

(*d*) Whether more than one joint was simultaneously or in rapid (*i.e.* a few days) succession attacked. Gout, gonorrhœal rheumatism, acute synovitis non-rheumatic, tubercle and Charcot are commonly, at any rate in first attacks, *monarthritic*. Rheumatism or rheumatoid arthritis is nearly sure to be at the bottom of a *primary polyarthritis*.

(*e*) Which joint or joints are affected. The proximal phalanx of the big toe is especially obnoxious to first attacks of gout, the knee to gonorrhœal rheumatism, the middle or proximal phalanges of the hand to rheumatoid arthritis, though of course exceptions are numerous, and the more so the greater the number of previous attacks.

(*f*) In cases where the patient is seen in a second (or higher multiple) attack, an equally careful account of the first one must be recorded if it can be possibly attained.

We may now consider the obverse side of the case, and briefly enumerate the main diagnostic points of each individual trouble, commencing with the most easily differentiated.

Charcot's Osteoarthropathy of Tabes.—Rapid (a few months or even weeks) onset of a nearly painless (so far as the joint itself is concerned, but lightning pains in the limbs very common) effusion into a joint, and excessive mobility of the articulation, should make a brief examination of the nervous system at once obvious to the mind. I am not aware that the joint changes are *ever the first and only signs of tabes*; loose bony outgrowths, and (or) atrophy and total disorganisation of the original articulatory surfaces are the principal anatomical changes. Some cases of rheumatoid arthritis so closely resemble in their local effects a Charcot's joint, that an idea is existent that the two diseases are alike in pathology; even granting a local identity, the remaining physical signs of tabes if present must always, I think, compel a diagnosis of Charcot, especially if only one (or two) joint be affected.

Flat Foot.—Requires to be mentioned in this connection because the patients so frequently come to us complaining of "rheumatism" in the ankle, and often pain up the calf of the leg. The patient will probably be a young and over-worked adolescent, and the pain will only be felt on or after long standing on the feet. Examination of the foot will show that the ankle is free from all signs of disease (heat, swelling, etc.), and that the pain is really in the scaphoid or head of the astragalus; the arch will be seen to be obviously fallen in—footprints (from a wet foot) may be taken to show this more accurately. A complete absence of pyrexia, or general constitutional symptoms, will practically, with the above, settle the diagnosis.

Gonorrhœal Rheumatism.—Is apparently in its essence a mild form of pyæmic joint. In its early stages will always, by the patient, and frequently by the medical man, be called rheumatism; in fact, I know of no pathognomonic sign in this stage to differentiate it, for the local processes are *then* identical. Later, it will be more likely to be thought of by (1) its obstinacy to remedies; (2) its confinement to one joint—usually the knee—without others being successively attacked during a rapid subsidence of the first ailing joint; (3) the implication of the outer structures of the joint, so that definite redness, slight œdema, and an angry, suppurative appearance supervenes; it is a curious fact that actual pus never does form in these cases. Once these phenomena have started suspicion, inquiry must be made as to a present or recent—the arthritis is commoner in the chronic stage of gleet—gonorrhœa or vaginal

discharge. In the absence of more serious signs of pyæmia, the above points will be sufficient to complete the diagnosis. We may tabulate the points of difference between simple and gonorrhœal rheumatism thus :—

Simple.	Gonorrhœal.
May or may not have a gonorrhœa or gleet ; the gonococcus is not protective against the rheumatic poison.	Certainly has either an active gonorrhœa or more probably a subsiding gleet.
Pyrexia smarter and nearly sure to be present.	Pyrexia may be absent, if present will be more irregular but persistent.
Polyarthritis, many joints recovering and getting worse coincidently or consecutively.	Probably a monarthritis, if a polyarthritis all the affected joints became implicated before any one of them got well.
If sweating at all it is profuse, and will go on all day ; smells sour.	If sweating at all it is that of pyæmia ; worse at night or on falling asleep.
Simple synovial effusion inside the joint.	Inflammatory affection of all tissues in and round the joint.

Traumatism.—In an acute attack the history is of course obvious ; but it must not be forgotten that slight (or severe) traumatism may be the starting-point of acute gout, or of any of the more chronic joint troubles ; and therefore when we are called upon to read the riddle of a chronic arthritic trouble we must not rest too satisfied with "traumatism," but must search the more carefully for a possible nervous (chronic degeneration) or constitutional (heredity, dietetic, toxæmic, etc.), element which has thus prominently exhibited itself in a damaged organ.

Hysterical Joint Complaints.—This is a comparatively rare form of neurosis, at least in a severe degree, found almost exclusively in the female sex, and in young adult life (say seventeen to thirty-seven), and more frequent in the knee than elsewhere. Its principal characteristic is that the patient complains most bitterly of the severe pain in the joint, but on examination there is not the slightest sign of anything being wrong with the structures (nor with the hip when the pain is in the knee)—no heat, no swelling, no grating ; it will then be found as an almost pathognomonic feature that the pain is just as severe on slight touch as when the joint is firmly handled, and that when the patient's attention is engrossed in some

other direction the pain on manipulation disappears. Frequently complete anæsthesia is required to eliminate some form of anchylosis, so resolute is the patient in preventing a movement, the slightest sign of which she finds exquisitely painful. If under an anæsthetic there is any effusion or grating, in fact anything pathological to touch, it is certainly not a case of pure neurosis.

Synovitis, Simple, Acute, and Chronic.—An acute synovitis with effusion, without any ostensible cause except local exposure, is described, though personally I have not seen a case. In its local manifestation, it is said to exactly resemble acute rheumatism, but without its general symptoms. A chronic synovitis is by no means so rare; it is usually started by some slight injury, and is probably maintained by continued use of the joint. It is characterised by being confined to one joint, associated with no general symptoms; it consists of a simple distension of the synovial cavity (possibly, especially in the knee, commencing with an external bursa); there is very little pain, except on excessive use of the joint. As it occurs in practice, the only difficulty in diagnosis arises when the distension has become so excessive as to loosen the articulatory ligaments, when it may be mistaken—especially as pain is so slight—for a Charcot's joint; the absence of any confirmatory signs of tabes should prevent mistakes.

Tubercle.—Offers many cases of doubt in the early stages, in fact, there is *at first* nothing pathognomonic about it. That this must be so is obvious if we bear in mind the morbid anatomy of the trouble. The arrival in a joint of a company of tubercle bacilli is not announced by anything more than microscopic changes of a very low degree of inflammatory character; in this stage it is a matter of minute cell changes which sound the alarm by twinges of pain. It is only when the granulomata have, by a growth and aggregation, reached a macroscopical size and bulk, that changes to the examining (from without) eye or finger are to be found. It may commence apparently either in the bone or synovial membrane. When in the former, we suspect its presence by the pain and starting in the joint being very much worse at night, and from the fact that this pain has arisen—in one joint only—spontaneously, or from traumatism so slight that there is very great discrepancy between cause and effect. When in the synovial membrane, there is as yet nothing to distinguish it from simple synovitis. As time goes on, however, we get suspicious, because this apparently trivial joint mischief will not subside; we look around, and find that the patient—probably from the first a frail-

looking object—is getting paler and thinner, inclined to mope, and capricious in appetite; we hear of consumption in near relatives; we notice that the joint is semi-voluntarily held in a constant fixed position — that of greatest ease — the slightest disturbance from which causes pain. By this time we have a strong conviction that tubercle is at the bottom of the mischief, but it is only when pulpy masses can be felt in the joint (in the knee or elbow), or an abscess forms (in hip, ankle, etc.), or the tissues external to the joint become chronically affected (wrist, *e.g.*) that this conviction gives place to *absolute certainty*.

Such is the common history of tubercular arthritis and of its diagnosis—let us hope that appropriate treatment will have begun with suspicion, not have waited for certainty. It must be remembered that now and again cases of fulminating tubercle, so to speak, are met with in which the joint very rapidly gets into a condition of acute panarthritis; here it is only the absence of any other plausible cause, and the persistency of great thickening long after the subsidence of acute inflammatory symptoms that will help us. For details of individual joints text-books of surgery must be consulted.

N.B.—There is one great caution to give in dealing with tubercular arthritis, and that is not to exclude tubercle because the patient is old; the disease is *very common* in elderly people in proportion to numbers alive.

Rheumatism.—Consists in its morbid anatomy of a simple synovitis, so far as the joints are concerned; and without other factors are at work, I believe, never *alone* gives rise to any other change. Typical acute or sub-acute rheumatic fever, with its pyrexia and sour sweats in adults (often, too, with endocarditis), or with its endo- or (and) pericarditis, or pleurisy, or history of chorea in children, consisting of a simple effusion, with pain running from joint to joint, some convalescent or well, while others are being attacked, cannot be mistaken for anything else. Its aberrant and chronic forms can better be discussed with differential diagnosis below.

Gout.—In the joints is essentially associated with, if not actually caused by, a deposit of urate of sodium in the articular cartilages. It, with its sudden onset during sleep, the intensity of the pain, the vivid red and shiny appearance of the joint, is equally difficult with rheumatic fever to confuse with any other trouble. For doubts and difficulties, *vide* below.

Rheumatic Gout. — The very existence of such a disease is denied by many, who dub it the refuge of the diagnostically desti-

tute. For myself, I certainly feel inclined to admit its existence, at least the existence of more than one factor producing the joints called rheumatic-gouty; though I must admit that many such cases are nothing but gout, and many are true rheumatoid arthritis (*vide* below).

Rheumatoid Arthritis, Osteoarthritis, better Chondroarthritis. — This is essentially at its commencement a *degenerative* proliferation of the cartilage cells, with an increase in the bulk of the articular cartilages, "the ruling passion—of reproduction—strong in death," but the result is poor stuff, with early decay and death stamped on it at its birth. There are two fairly marked types, (1) with (2), without marked inflammatory reaction in the other structures of the joint, especially of the synovial membrane. The former is mainly found when the disease attacks young people (it is most important to remember that this affection is, aye frequently, seen in young people, even in children); the latter is the commoner type in elderly and old patients, though here, too, it may have been preceded by attacks of the more inflammatory type. The principal characteristics of both forms—when the acute features have passed away in the one, and under ordinary circumstances in the other—are (1) the lipping of the cartilages, so that the outlines of the joint are felt in bolder relief than usual; (2) grating in the joint; (3) stiffness and diminished mobility; (4) frequently, and especially in the metacarpo-phalangeal joints, a subluxation of the articulation, so that the distal bones are diverted from their natural direction.

Differential Diagnosis of the Last Four Affections, viz. Rheumatism (Acute and Chronic), Gout, Rheumatic Gout, and Osteoarthritis

We may consider this as it occurs in the several epochs of the affections :—

1. On the post-mortem table.
2. After treatment has been tried for a little while.
3. During a first attack.
4. In a period of quiescence after a first attack.
5. In subsequent attacks.

1. **On the post-mortem table.**—This method of diagnosis comes in very late for purposes of clinical medicine, and is only occasionally able to give us information which is of use in estimating the

morbid condition in the earlier affections of other patients. The reasons why it is of such comparative uselessness are: (1) that the troubles under consideration are practically never fatal (a few cases of rheumatic fever must be excepted) in their earlier stages, when commencements of morbid changes could be usefully studied; (2) that when the advanced conditions are found, after death from other diseases, the history of the joint complaints has been unrecorded or lost from the overwhelming interest or importance of the later disease. However, the facts are very simple, and diagnosis thus made is easy enough:—

If urate of sodium be present in streaks or patches on the cartilages, we know for a certainty that gout has been at work, either alone or with other factors.

If the cartilaginous coverings of the bone inside the joint are worn away, and the bare bony surfaces are eburnated or polished, and there are no traces of urates about, we know for a certainty that osteoarthritis has been the disease.

If both the above changes, viz. urates and eburnation can be seen, we are justified, I think, in assuming that the morbid physiology of both, *i.e.* gout and rheumatoid arthritis, diseases has existed, and the more shall we think that osteoarthritis has been present the larger the superficial area of the end of the bone, this being the final representation of the pathological growth of the cartilage at the edges of the articulation—the lipping to be felt during life.

If the disease has been a pure rheumatism—even chronic—we shall find very little or no organic change. In the acute affection there will be but clear fluid in the joint, with very little (probably none) congestion of the synovial fringes. In the more chronic condition of rheumatism there may be a little stiffening of the ligamentous structures of the joint, difficult to appreciate, and it is, I think, here possible to meet with a slight loss of cartilage, suggestive of osteoarthritic changes, for I believe that the frequently repeated onslaughts of pure rheumatism may by their local influence initiate (? carry to their final stages) those changes which in osteoarthritis are started and carried on by loss of trophic nerve control.

[If the cartilages are gone, the bone bare but not eburnated, rather cancellated (rarefactive *v.* sclerosing osteitis), and loose bits of bone are found, and the joint flail-like, we infer that it is a Charcot's joint; though in dried specimens without any history we must not forget the probable identity of cause (*i.e.* loss of trophic influence) between this and osteoarthritis, and therefore the

possible identity of result; though I think a true Charcot never has eburnation.]

2. **Inferences from Treatment.**—This method again may be looked down upon as coming a little late in the day; but we may urge that as medicine is not, and can never be (*pace* the scientific pharmacologists and physiologists) an exact science, and as therefore treatment often has to be started on empirical supposition, it is but fair to allow us to gather information as to diagnosis as we proceed; and to use the influence of one form of treatment over a joint affection as a starting-point for endeavours to make a complete deduction as to the nature of such affection. Salicylate of sodium is the drug above all others which is now likely to be tried in all doubtful cases of joint trouble, and so great is the influence of this drug (amounting almost to a genuine specific) over pure rheumatism, that it has become a nearly fixed article of medical faith that "the greater the relief afforded to pain, pyrexia, and general discomfort, the greater the share of rheumatism in the production of the trouble." If the relief be speedy and complete, the diagnosis of pure rheumatism is assured; if relief be not at least very marked, we at once suspect, no matter what the age of the patient (I have already noted the frequency of acute symptoms of rheumatoid arthritis in young subjects), that there are factors in the trouble which are likely to lead to permanent changes in the joint, and consequent crippling; we must carefully search for a gonorrhœa or gleet, and inquire more anxiously into family history. There is one important exception to be made to this usual rule of the influence of salicylates over rheumatism, viz. that if rheumatism attacks an individual who has had a long or severe drain on his constitutional strength, we find salicylates much diminished in utility or even quite useless; I have met with such cases in women after prolonged lactation, in adolescents who have been poorly fed for years, and I suspect that in the rheumatism so common after scarlet fever the same conditions may hold.

3. **During a First Attack.**—This is naturally the time when diagnosis is most required; more perhaps for the sake of our own reputations as prophets, and for the advice we are to give as to warding off future or progressive trouble, than for the immediate sake of the patient, for whom any sensible treatment is little likely to be at once disastrous. The previous paragraphs on symptoms and pathology have already indicated the directions in which diagnostic aid is to be sought, but for the sake of emphasis we may here tabulate some of the cases, and offer a few general remarks.

In young subjects—say under twenty-five—gout is so rare that unless it be a very typical case—nocturnal onset, one joint, intensely painful, very shiny and red—or unless there be a very strong and direct family history of gout, this disease may be excluded, and the problem—except for tubercle (*vide* p. 237)—becomes simple rheumatism *v.* rheumatoid arthritis.

They are alike in—

1. Many (and any) joints are simultaneously or in the same attack affected.
2. There is often effusion; it is in the rheumatoid arthritis of young subjects that effusion is commoner, and that with pyrexia and general symptoms.
3. The trouble *appears* to be a synovitis.
4. There is no grating in a first attack.

They differ in—

True Rheumatism.	Rheumatoid Arthritis.
Cardiac bruits and serous inflammation very common; more common the younger the patient.	Cardiac bruits have no known connection with the affection, and if present have had a previous origin.
A sequence of joints very commonly affected, so that different stages are found in the same case.	Sequence of joints not well marked; usually many at the same time, especially if the small joints are attacked; very often only one or two larger joints affected.
Shape of joint pretty natural, except for effusion into its cavity.	Shape of joint a peculiar spindle, as though atrophy above and below, and the joint enlarged in the middle; especially noticeable in phalangeal articulations. This probably means that there is a slow outgrowth of cartilage before the symptoms, possibly also a slight atrophy of structures above and below the joint.
Sweating very common, and of a peculiar sour smell; this is less marked the younger the patient.	Sweating not a marked feature, and if present has no sour smell.
Fibrous nodules occasionally seen in fibrous tissue about a joint.	Fibrous nodules not seen.

True Rheumatism.	Rheumatoid Arthritis.
Pyrexia and heat of skin usually marked.	Slight pyrexia may be present though not common; skin in general is certainly not hot to the touch.

In middle age, or rather from twenty-five up to say forty-five, true gout becomes more frequent as the cause of a first attack, and according to my experience rheumatoid arthritis is less frequent, at least until thirty-five be past, when it again has an increase. It is between twenty-five and forty-five that I believe chronic rheumatism—a form without much pyrexia or swelling, only a little pain and stiffness—is a very prevalent affection; though I admit it is very difficult to draw a hard and fast line between this affection and rheumatoid arthritis (*vide* Diathesis below). In old age, say from fifty-five upwards, I believe that first attacks of gout again become rarer, and that, too, in proportion to age, while (tubercle and) atrophic changes of an osteoarthritic or crippling character become the prevailing affection.

4. **In a Period of Quiescence after a First Attack.**—It is in this period that we have to look out for stiffness, grating, or deformity as evidence of the local trouble, and general symptoms that can be attributed to the constitutional affection of which the arthritic attack may have been only an outward manifestation.

True rheumatism will have left practically no objective evidence of its presence; a little stiffness and slight pain that gets less and less are its only sequelæ. It may be, however, that slight pyrexial outbreaks, with a little local joint trouble, remind the patient that his disease is scotched only, and not killed; and these are the cases which, in my opinion, have a tendency to eventuate in crippling osteoarthritis as age creeps on. These, too, are the cases which in *young* people are so affected by changes in the weather. I believe that when a young person admits himself a living barometer he is the subject of true chronic rheumatism, which in his later years is likely to become crippling. Pleurisy, pericarditis, endocarditis, occurring on very slight or no provocation, will also throw light on the rheumatic nature of a previously doubtful joint attack.

If the attack has been acute rheumatoid arthritis, the stiffness is never likely to so completely disappear; a grating and deformity, with possible subluxation of joint, will probably soon make their appearance. On true rheumatoid arthritis my experience is that

in *young* subjects weather has no influence; it is only when age creeps on that such appears to be the case, but then I believe it is more due to the effect on senile tissues in general than on the joints in particular, and hence an elderly living barometer does not thereby tell us much to aid differential diagnosis. Beyond the obstinacy to treatment, and the local changes, there are no constitutional sequelæ and complications.

If the attack has been modified (typical will have been easily recognised) gout, then, as in epileptics, the patient will probably express himself as feeling very much better in general health than before the attack. Tophi must be carefully searched for, and the urine should be examined pretty frequently for any excess of uric acid or diminution in total nitrogenous output, hinted at by a low specific gravity.

5. In Subsequent Attacks.—But little remains to be said under this head. If the history of the first attack (*q.v.*) be not available, the evidence from all sides—family, constitutional, and local—will have been steadily accumulating, and, though some features may still remain obscure, the main points—the tophi and urine of gout, the cartilaginous outgrowth of osteoarthritis, the morbus cordis with but slight local arthritic damage of rheumatism—will be almost sure to point only too clearly to the correct diagnosis.

THE ARTHRITIC DIATHESIS

Personally, I am indebted to the writings of Mr. J. Hutchinson for this expression; that he was the first to utter it, is not likely, for the idea conveyed by it must have been present, one might say, for centuries, in the mind of the profession. Whatever its origin, I accept it as clearing up and harmonising many of the doubts and difficulties in arthritic diagnosis.

I will first define precisely what I mean by the expression. It is this: that there are certain people who, owing to the intrinsic quality either of the joint structures (bones, ligaments, cartilages, and synovial membranes) themselves, or of the nerves which undoubtedly govern their nutrition, find themselves in this position, —when from exposure, diet, or other unfavourable environment, a blood dyscrasia or other constitutionally morbid state arises of such a nature that some tissues are likely to suffer from imperfect nutrition; the joint tissues above mentioned, or their nerves, are the parts of the organism upon which the stress will first fall: they are

the structures to suffer. Furthermore, when once such a morbid local process has been thus started in the joints, there is in these people (1) a greater tendency than in others for the process to extend; (2) a probability that it will in this extension lose any possible *specific* character it may have originally possessed—gouty, rheumatic, etc. — and become generally and indiscriminately degenerative and destructive; (3) a greatly diminished and less ready capability of repair if, and when, the constitutional dyscrasia has disappeared. Finally, to complete the story, I believe that though in the main such joint vulnerability is an inborn or hereditary—it may be from fairly remote ancestry—property of the individual, it may be acquired by personal habits and total environment (*vide* p. 7), and then in some degree (? crescendo) transmitted, but this transmission need not be an accurate and identical one, just as an epileptic need not necessarily have an epileptic child, but one that is generally weak in the nervous system; so I believe a *gouty or rheumatic* ancestor may have *generally* arthritic descendants.

These generalised laws or propositions which have been framed not by deduction—from few—but by induction—from many cases —constitute the explanation of the expression "crippling changes" which I have several times used, intending thereby to represent the *generalised* destructive processes which proceed from *originally specialised* causes. They explain the identity of the pathological anatomy of a Charcot's joint with that of an advanced osteoarthritic one, and of the latter with that of a chronic rheumatic joint in which the diseased process has overstepped its natural boundary of the synovial membrane, and led to destruction of ligaments, etc.; and this ultimate anatomical identity proves how impossible it is in chronic joint troubles to be certain that simple synovial rheumatism will not, *clinically*, in the long run assume the features of rheumatoid arthritis.

They possibly give us a clue to the reason why gonorrhœal rheumatism is so rare compared with a urethral discharge, and why tubercular joints are much less frequent than phthisis.

Their bearing on the family history of a case of joint affection is important, but not decisive; they explain at once the "why," but they do not directly give us the "how," of a particular attack. There can be no doubt about the influence on diagnosis of a definite history of rheumatic fever, or of gout, in immediate ancestors or very close collaterals; but when we hear that a father, mother, uncle, aunt, or grandparent had suffered *at an advanced age* from rheumatic gout, the specific indication is very much weakened, and

we are thrown more completely on to local signs for the precise diagnosis; a creaking, stiffened joint, a small tophus in the ear or elsewhere, a cardiac bruit, etc. In conclusion, let me emphasise a final statement—because or when we can find a hint of this predisposition we must not rest content with it, we must be all the more anxious to find the precise local factor in its earliest stages; for it is only in these that treatment is even likely to be curative. When destruction is widespread and irreparable, precise diagnosis may be interesting scientifically, but it is of precious little use to our patients, who must ever be the first care in our minds.

CHAPTER VIII

DISEASES OF THE NERVOUS SYSTEM

SECTION I.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

BEFORE the problems of regional and pathological diagnosis of nervous affections can be intelligently discussed or tabulated, it is absolutely essential to sketch in slight detail the anatomy and connections of nerve structures, and the functions they are assumed or proved to possess.

Recent researches into the microscopic anatomy of the nervous system have rendered it very probable, if not actually proved, that all nerve structures, without exception, from end bulbs or taste corpuscles to cerebral hemispheres, are built up of neurons, and that these neurons or nerve units are all formed on the same structural plan, varying only in details of size of cells, length and number of processes, etc., and of course in physiological function. Each neuron, then, consists of:—

(1) *A Cell with Large Nucleus.*—The substance of the cell shows a very fine fibrillar constitution with minute granules between the fibrillæ. The body of this cell gives off—

(2) *Numerous Processes*, all of which ultimately end in dendrites, and one at least of them becomes the axis cylinder of a nerve of ordinary anatomy; this latter is spoken of as the neuraxon.

(3) *Dendrites.*—An expression used to designate the extremely fine fibrillulæ which form the final endings of all the processes of the neuron cell. It is by the interlacing of these dendrites—aptly termed by Foster a synapsis—that separate neurons are brought into physiological or functional connection with one another, though it must be clearly understood that no structural or anatomical continuity has been proved to exist between the dendrites of different neurons; in fact, the assumption is that dendrite acts on dendrite

in a manner analogous to that of the action of the primary and secondary coils in an induction electric machine.

The terminals of sensory organs and the end plates of motor nerves in muscles are probably nothing more than neurons of very abbreviated dimensions. With these exceptions, and possibly some in the cord and brain, all neurons possess processes of measureable length, and the neuraxons may be (as in the nerves of the extremities) many inches long; but, except for this variation in length of the cell processes, they are all identical in structure, and collectively build up the whole nervous system. Accepting this as the anatomical unit, we may now proceed to consider how these units are combined.

TABULAR VIEW OF THE ANATOMY OF THE NERVOUS SYSTEM

A. *End Organs or Terminals*, each probably a very abbreviated neuron :—

1. In muscles intervening in
   - Contraction and Relaxation.
     - Voluntary muscles under control of will.
     - Involuntary muscle fibres of heart, blood-vessels, viscera, etc.
   - Sensation of nature and amount of contraction and relaxation of voluntary muscles = muscle sense. If such sensory structures exist in involuntary muscle they produce in health no percept on the general sensorium.
2. In skin reacting to . . .
   - Touch.
   - Variations in temperature.
   - Pain.
   - All other cutical stimuli.
3. In special sense organs subserving . . . . .
   - Sight.
   - Hearing.
   - Taste.
   - Smell.
4. In glands and membranes governing . . . . .
   - Absorption.
   - Excretion.
5. In all other tissues of the body superintending and regulating
   - Life,
   - Growth, and
   - Decay.

B. *The Peripheral Nerves.*—These are the main trunks (neuraxons and collaterals) of the neurons; they possess dendritic

endings, and synapses with the end organs; they conduct stimuli or impressions to and from the end organs, thus constituting telegraph wires between the brain and cord and every part of the body. The exact anatomical relationship and distribution of the peripheral nerves constitute a branch of ordinary descriptive anatomy, an accurate and reliable knowledge of which is essential for the finer minutiæ of the differential diagnosis of peripheral (and other) lesions. A detailed description would in this book be out of place.

C. *The Spinal Cord.*—This constitutes a collection and primary arrangement of the connections between the brain and the periphery. A transverse section (or longitudinal one) accordingly presents a picture composed, so far as nervous elements are concerned, of certain groups of cells and bundles of fibres all cut in various sections according to their direction. Those groups and bundles, which are tolerably well known and understood, may be thus tabulated:—

CELLS OF THE CORD

One process of each of which forms the neuraxon or axis cylinder of the fibres of

In anterior cornua (the main bulk of them, at any rate).	Anterior roots or peripheral motor nerves, possibly also of vasomotor nerves.
In Clark's column.	Nerves to and from viscera, and to and from direct cerebellar tract.
Others scattered through the gray matter, and possibly some of anterior cornua.	Neurons or nerve-tracts, with origin and dendritic terminals totally within the cord.

FIBRES OF CORD

Which are the neuraxons (or collaterals) of neurons, the cells of which lie in

Posterior median column. Posterior external ,, Posterior roots.	The ganglion on the posterior root of peripheral nerves.
Lateral pyramidal tracts. Direct ,, ,,	Rolandic or motor area of cerebral cortex.
Ascending antero-lateral tract of Gowers.	The reticular formation of medulla and pons, and also in the ordinary gray matter of cord, as intracordal neurons.
Anterior roots.	Anterior cornua.
Direct cerebellar tract.	Clark's column, and probably also cerebral and cerebellar cortex.
Many unnamed fibres, both in white and gray matter.	In gray matter = intracordal neurons.

D. *The Medulla Oblongata.*—Structurally, this is the continuation upwards of the cord, but the arrangement of its neuron cells and stems is more complicated and intricate. Only the more important (as far as our present knowledge is concerned) and apparently simple ones are here tabulated :—

Cells of the Medulla

Groups just below the floor of the fourth ventricle in serial continuation of those of the anterior cornua.	One process of each of which forms the neuraxon or axis cylinder of a fibre of	Twelfth nerve, or hypoglossal. Eleventh nerve, or spinal accessory. Tenth nerve, or pneumogastric Ninth nerve, or glosso-pharyngeal. Eighth nerve, or auditory.
Groups in Funiculus gracilis. ,, cuneatus. ,, rolandi.		Tracts to cortex of cerebellum and cerebrum, to the former by the restiform body, to the latter through the reticular formation and fillet.
Groups scattered through the reticular formation.		Antero - lateral ascending tract. Posterior columns.

Fibres of the Medulla

Pyramidal tracts.	Which are the main trunks of neurons starting in the cells of	Motor cortex of brain.
Funiculus gracilis. ,, cuneatus. ,, rolandi.		Posterior root ganglion, and also (above the cells of the funiculi) cells of cortex of cerebrum, and cerebellum.
Direct cerebellar tract.		Clark's column, and also cerebellar cortex.
Cranial nerves from sixth to twelfth.		Cells in floor of fourth ventricle.

E. *The Pons.*—The main bulk of the superficially visible fibres run in a transverse direction ; the deeper parts are really the continuation upwards of the medulla. The cells and fibres most essentially pontine are the following :—

Cells of Pons

Groups round the central canal.	A special process of which forms the axis cylinders of	Fourth nerve. Third „ (chiefoculo-motor).
Groups in the roof.		Going to cerebrum and cerebellum.

Fibres of Pons

Transverse.	Which are the main trunks of neurons starting in cells of	Cerebellum.
Oblique.		Cerebrum and cerebellum.
Vertical or pyramidal.		Motor cortex of brain.
Transverse and oblique, passing in all directions.		Probably scattered through the whole cortex of cerebrum and cerebellum and formatio reticularis, and also basal ganglia.

F. *Cerebrum and Cerebellum.*—In these organs the connections and arrangements of neurons attain their highest degree of complexity and obscurity. Some few we think we know, but of the vast majority our knowledge is practically nil. In the section on cerebral localisation more details of the anatomy and functions of the brain will have to be given. For our present purposes the following table will be sufficient:—

Cells of Cerebrum and Cerebellum

Ascending frontal parietal and rest of motor cortex so called.	A special process of each of which forms	Corona radiata, anterior two-thirds of posterior limb of internal capsule and pyramidal tracts.
Frontal lobes.		Fibres running antero-posteriorly to opposite cerebellar lobes, and to the rest of cortex cerebri also.

FIBRES OF CEREBRUM AND CEREBELLUM

Of frontal, occipital, and temporo-sphenoidal convolutions.

of motor convolutions.
of internal capsule.
of anterior limb.
of posterior ,, anterior two-thirds.
of ,, ,, posterior one-third.

} Main trunks of neurons starting in cells of

Convolutions of same side and of opposite side (through corpus callosum).
Convolutions of cerebellum.
Gray matter of cortex.

} Frontal cortex.
Motor cortex.
Occipital and temporo-sphenoidal cortex.

Note.—Between the anterior and posterior roots of the spinal cord on the one hand, and the cerebral hemispheres on the other, there is a total decussation of impulses, if not of actual fibres. But such functions and anatomical connections as the cerebellum exerts on, and possesses with, the cord are apparently not crossed.

After this brief dogmatic sketch of the structural connections of the nervous system, we must proceed to a similar tabulation of the—

FUNCTIONS OF NERVE MATTER

We will commence as before with THE NEURON.

1. *The Cell.*—While there is still some dispute as to whether the cell possesses any function of a transforming or reinforcing character, there is unanimity in accepting a purely nutritive function as the chief, if not the only, function properly belonging to the cell. At any rate, it is a clinical fact that neuraxons, collaterals, and dendrites, and consequently the synapses of the dendrites, all undergo complete degeneration and death when separated from the neuron cell, or when the neuron cell itself is killed; and nutritive disturbance of the cell is rapidly followed by similar changes in the other parts of a neuron, and structures (non-nervous, *e.g.* muscles) to which the nerves are distributed.

2. *Neuraxons and Collaterals.*—The only ascertained function of these parts of a neuron is to transmit unchanged along their length impulses derived, or started (*a*) from sources external to the body, including in this term food in the alimentary canal, and air and its impurities in the air passages, as well as stimuli applied to the skin,

etc., (*b*) from the synapses with dendrites of other neurons. They are thus purely and absolutely analogous to telegraph wires, carrying to and from every structure of the body messages which result in the performance of every possible physiological process, whether motion, perception, nutrition, secretion, etc.

3. *Dendrites, and Synapses of Dendrites.*—It is by these we now assume that impulses are transmitted from neuron to neuron, and transformed in character. It would seem as though the synapses afforded an opportunity for a sort of inductive action of neuron on neuron; the actual resultant of any impulse from any source depending upon the inherent qualities of the neuron brought into induced action and its connections (*a*) with other neurons to which the impulse may be again transmitted, or (*b*) with terminal organs, muscle, gland cell, or other tissue capable of exhibiting a final resultant.

Peripheral Nerves.—As these are simply neuron complexes, their functions in gross are those of their units; in detail they carry the impulses which eventuate in—

A.—*Movements in Voluntary Muscles which*—

In Health.	In Disease.
Are possible to an amount known by experience to each individual; are under the control of the will, and capable of co-ordination for any purpose.	Are weakened, or absent, or incoordinated.
	In volition { Time or order. / Amount.
	Involuntary spasms { Tonic. / Clonic.

B.—*Sensory Perceptions*—

A known experience of the individual, differing materially in different individuals.	Excessive / Weakened / Lost / Delayed / Perverted	in relation to	Temperature, / Pain, / Touch, etc.

C.—*Reflex Processes*, any of which may incidentally affect consciousness and become a percept. They are divisible into three groups:—

Group I.—Experimental, or acting through cutaneous external stimuli:—

Superficial—

	In Health.	In Disease.
Plantar, cremasteric, etc.	Present, but somewhat variable in amount.	Obviously increased. ,, decreased. Absent.

Deep—

Knee jerk. Elbow jerk, etc.	Present, but also somewhat variable.	Increased, diminished, absent.

Group II.—Natural from the viscera with contents or condition for stimulus, and involuntary movement or secretion, etc., as the resultant. Of these there must be very many, but in only a few cases and conditions are of much use to us clinically:—

	In Health.	In Disease.
Those of bladder. ,, rectum.	Co-ordinated for purposes of voluntary micturition and defæcation.	Inco-ordinated, and often removed from voluntary control.

Group III.—Vasomotor regulating the supply of blood to a part—

In Health.	In Disease.
The essential point is the local control over local blood supply.	This local control is frequently lost or perverted.

D. *Trophic Influences.*—Clinical evidence, as well as experimental, tends to suggest that, besides the above noted trophic influence of the neuron cell on its own processes, there are influences constantly ascending and descending peripheral nerves which are essential for the proper nutrition, growth and decay of the structures which, in their ordinary functions, are under the control of the nerves distributed to them—muscles, for instance, or sensory end organs, glands, etc.—and there is a certain amount of evidence to show that these influences are something active, liable to excess or perversion, as well as absence, for it is found that irritation or inflammation of nerve structures is more effectual in producing destruction or degenerative effects than is mere section of a nerve trunk. Illustrations of trophic changes will be noted presently (*vide* p. 266).

E.—*Special Sense Nerve Phenomena and Mental Processes.*—These

will be noted when the brain is under consideration. We may here merely mention—

Sight, Hearing, Taste, Smell	All varying in acuity in health.	And in disease may be increased, perverted, or diminished.

Spinal Cord.—To enumerate in detail the functions of the cord would in large measure be to repeat what we have said on peripheral nerves. Its cells are constantly engaged in the task of superintending the nutrition of peripheral nerves and other structures, as well as of intracordal fibres or neurons, as has already been noted. The principal function of the cord, as a whole, is to act as a sort of subordinate office to the brain's head office, and by its complexes of neurons to arrange and co-ordinate impulses passing through it. Thus it is the final manager of efferent impulses to voluntary muscles arranging their order; it is the primary manager of afferent impressions, enabling our minds to become precisely and accurately conscious of our environment; and lastly, it possesses innumerable mechanisms for the performance of reflex functions of all descriptions: and these may (*a*) be performed entirely by the cord without notification to the sensorium, or (*b*) cause such notification so that we perceive the reflex, but (*c*) they are always liable to control from the head office of the brain under all circumstances.

Brain, or Encephalon.—As the seat of what is termed the mind, the encephalon remains the supreme governor and controller of every process going on in the body. It is the head office to which all information from every part must be sent, whether we are conscious of such information or not, and it retains to itself the power to intervene in and control everything, but allows generally to the cord a very large measure of home rule in those processes which are mainly concerned in the mere sustentation of the animal framework.

Having thus considered anatomical structure and physiological function as separate entities, we have now to associate the two into mechanisms for purposes. This, even in the peripheral nerves, is not always a matter of mathematical precision; in the cord the difficulties become much greater, while in the brain they are as yet insurmountable except in a few special cases, and in a most general manner. We shall arrive at the most available certainty with greatest clearness by taking functions and tracing their path-

ways; the following tables are apparently reliable as far as our present knowledge goes, but being based upon clinical experience and experiment, are liable to some alteration as our knowledge grows in exactitude.

Motor Tracts or Pathways of Voluntary Muscular Movements

The nerve impulses are centrifugal; so, taking the same course, we have—

1. Highest volitional, or will centres of doubtful locality, probably (according to Hughlings Jackson) the cortex of the frontal lobes.
2. Rolandic region of cortex.
3. Corona radiata.
4. Anterior two-thirds of posterior limb of internal capsule.
5. Pyramidal tracts—
   - (*a*) In crura cerebri.
   - (*b*) In pons.
   - (*c*) In medulla (including the decussation).
   - (*d*) In cord (crossed and direct).
6. Cells of anterior cornua.
7. Peripheral motor nerves.
8. End plates.
9. Muscles.

In any given voluntary movement the parts played by these various structures are as follows:—

In (1).—The wish starts by psychological processes or influences from without, and takes a less amorphic shape as nerve impulses, which pass along the processes of the neurons concerned; by dendritic synapses these impulses are transmitted or passed on to the neurons of the rolandic region.

In (2).—These physical impulses are marshalled and co-ordinated in a manner that may be compared to the first sorting of letters at the General Post Office into those for each sub-district or "route," and just as by arrangement letters can be sent by an alternative route, when one is blocked, with slight resultant confusion in their delivery, so by the dendritic synapses of innumerable cortical neurons can these impulses be transmitted by other than the usual path, with resultant delay or disturbance in movement. Herein lies

the commencement of the explanation of the common statement that "Movements and not muscles are represented in the cerebral cortex (and cord)." Each neuron of the rolandic cortex has to share in many movements, but never *completely* causes one; hence *slight* cortical damage is likely to impair many movements, but not absolutely to paralyse any.

From (2) impulses pass through (3), (4), and (5) in a comparatively simple manner along the neuraxons, and break on the complicated synapses of their terminals with the dendritic commencements of (6).

In (6), with its connections, goes on the final sorting of impulses for delivery along the neuraxons of the appropriate members of (7); thus is completed the explanation of the statement inserted above relative to movements rather than muscles being represented in brain and cord.

Along (7) messages are carried again in a very simple manner to reach (8) and (9), and produce in them appropriate reaction.

For clinical diagnosis it is important to note the absolute independence of the nutrition and life of the neurons of (1), or level A, of (2) to (5), or level B, and of (6) to (9), or level C. A lesion of destructive character in level A leaves the mechanisms of levels B and C, *qua* a piece of machinery, quite untouched and ready to start and carry out co-ordinate movements, provided only that impulses can reach them somehow; and, similarly, B and C are nutritionally quite independent; hence we find at the bedside that a paralysis due to lesion in level C is characterised by the flaccidity with rapid wasting of the affected muscles, and probably other trophic disturbances of the skin and its annexes. A paralysis due to a lesion in levels B or A is characterised by retention of the tone and of volume of the affected muscles, and absence of trophic disturbance of other structures.

Sensory Tracts or Pathways of Afferent Impressions from without

These impulses are centripetal, and hence we begin with—

1. Peripheral sensitised end organs in skin, muscle, or special sense mechanisms (these are the best understood clinically and microscopically, but analogous nerve structures must exist in every tissue and gland of the body).
2. Sensory nerves of descriptive anatomy.

3. Cells in the ganglia on the posterior roots.
4. Posterior root of spinal nerve.
5. Posterior columns of cord, external and internal, and probably also Gowers' antero-lateral tract, and the direct cerebellar tracts.
6. Cells in funiculi gracilis, cuneatus, and rolandi, and also Clarke's column.
7. Cells and fibres in formatio reticularis and fillet of medulla and pons, tegmentum of crus cerebri, inferior peduncle of cerebellum, and the cortex of both cerebrum and cerebellum.

With regard to the part played by these various structures in the perception of an external stimulus, we are very much more in the dark than is the case with the corresponding motor tracts; but the following statements would appear to be justified:—

In (1) the external impulse, whether of touch, temperature, pain, etc., is received and converted into what we can only call a common nerve impulse, which is then carried by way of (2), (4), and (5), as along telegraph wires, until it breaks on the dendritic synapses of the terminals of these, and thus can impress itself in some way on the terminals of the neurons in (6) and (7). It is commonly assumed and universally accepted as a legitimate deduction from experiment and clinical evidence that Nos. 1, 2, 3, 4, and 5 form a lowest level or neuron complex, corresponding to the lowest level on the motor side, and that this lowest level is dependent for its nutrition on No. 3; the injury or destruction of which results in corresponding degeneration or destruction of the whole level. But after we leave this level precise knowledge ceases, and speculation reigns as to the number of serial neuron levels and as to the exact paths by which impulses reach the sensorium, and even as to the locality of the sensorium. And, again, as regards co-ordination of, or judgments upon external impressions, we only know that disease of the lowest level (as in tabes dorsalis) leads to great perversion of them; *e.g.* a touch is referred to a totally wrong spot, but we do not know whether this lowest level is the only co-ordinator of afferent impressions, or even if it is the chief one.

Reflex Paths

So far as is known, there are no special paths reserved entirely for reflexes; the impulses travel by the ordinary sensory and motor

nerves. We may, however, discuss a few points in this connection. For a reflex phenomenon we require a chain of structures which we may thus tabulate :—

A sentient receptive surface or end organ on which the stimulus may act.	Skin or external surface in the experimental class. Mucous membrane or epithelial cells in visceral class. Tissue elements (?) in vasomotor class.
An afferent nerve.	The ordinary afferent nerves of the organ or area in question.
A central connection of afferent and efferent mechanisms.	This, so far as we can judge, is not an organic or structural connection, but merely the functional synapses of the dendritic terminals of the respective sensory and motor neurons in the central system, allowing a transmission of impulse.
An efferent nerve.	The motor nerves of voluntary or involuntary muscles, corresponding to the class of reflex in question ; and nerves carrying efferent impulses for other organs.
A terminal organ to exhibit the reflex, whatever its nature.	Voluntary muscles in Class I. Involuntary muscles, fibres, and epithelial cells of glands of viscera in Class II. Involuntary muscle fibre in arterial coats in Class III.

For the reflex to take place it is essential that this chain should be complete in each link, and that there should be no strong inhibitory influence at work capable of stopping the reflex.

Class A.—The true or superficial reflexes may occur from any area of skin or from the conjunctiva. Those most commonly looked for are :—

Plantar,	involving	health of	1, 2, and 3 sacral nerves	and	connections.
Cremasteric	„	„	1, 2, and 3 lumbar	„	„
Abdominal	„	„	8 to 12 dorsal and 1 lumbar	„	„
Thoracic	„	„	1 to 8 dorsal	„	„
Conjunctival	„	„	5 and 7 cranial	„	„

but in special cases many others are looked for as indications of health or disease of specific nerves and segment of the cord.

Of the so-called deep or tendon reflexes at knee, elbow, wrist, etc., we know that the path for them must, in some of its details, be different from that of the superficial ones, for the two

can vary independently of one another in degree; the knee jerk, for example, is often absent, while the superficial reflexes of the leg are still obtainable. The knee jerk has been more fully studied than any of them, and about it we may make the following statements, with deductions:—

It requires for its production—

(1) A certain degree of myotatic irritability (Gowers) in the quadriceps extensor muscle.
(2) The integrity of the third and fourth lumbar nerves, and their synapses in the cord.

It is capable of being modified by influences from the encephalon, which influences are found to travel by the pyramidal tracts of fibres, and are believed to be of two kinds: (*a*) restraining from the cerebrum; (*b*) reinforcing from the cerebellum (Jackson).

As deductions from these premises—

Increased knee jerk means	(*a*) Diminished cerebral control. (*b*) Increased cerebellar influence. (*c*) Increased irritability in some link of the spinal and peripheral chain.
Diminished or absent knee jerk means	(*a*) Increased cerebral control. (*b*) Diminished cerebellar influence. (*c*) Diminished irritability (or break) in some link of the spinal and peripheral chain.

When the knee jerk is found to be materially altered in character from an average expected normal, we should try and estimate which of these influences is at work. The common clinical examples are:—

		Indicated by
Knee jerks increased from cause (*a*).	Paralysing lesion of rolandic cortex, or internal capsule.	Apoplectic seizure of ordinary type.
	Lateral sclerosis primary.	Chronic history of increasing weakness and jerkiness in walking.
	Old transverse myelitis above lumbar enlargement, causing, *e.g.*, lateral sclerosis of lower part of cord.	History of acute paraplegia.

		Indicated by
From cause (*b*).	Occasionally apparently in cerebellar tumour, causing more irritation than destruction.	Headache, vomiting, and optic neuritis.
From cause (*c*).	Peripheral neuritis in early stages.	Pins and needles, and weakness.
Knee jerk diminished or absent from cause (*a*).	Occasionally apparently in tumour of cerebrum.	Headache, vomiting, optic neuritis, etc., Jacksonian epilepsy.
	Meningitis, vertical and even basic.	Pyrexia with cerebral symptoms.
	In shock their absence is probably partly thus explained; general paralysis of insane in later stages.	Mental features.
From (*b*).	Occasionally apparently in abscess and tumours that have destroyed some parts of cerebellum.	As above.
From (*c*).	Peripheral neuritis, later stages, acute or chronic anterior poliomyelitis.	Rapid or chronic wasting of muscles with R.D.
	Tabes dorsalis.	Lightning pains; Argyll Robertson pupils, etc.
	Pseudo - hypertrophic paralysis.	Method of getting up from the floor.
	Myelitis of lumbar region.	Acute paraplegia.

In some of the above cases the meaning of the alteration is fairly clear and definite, but it must be confessed that in tumours and in inflammation within the cranium neither the result nor its cause is always very obvious. In epilepsy, again, the frequent absence, and in general paralysis of the insane the frequent exaggeration, are not at once readily explicable; in hysteria the knee jerks are very variable, and I have, I believe, found them absent in an otherwise perfectly healthy individual.

Class B.—Visceral. For the bladder and rectum (it would seem probable also that the rest of the alimentary tract and other

viscera are under similar control by means of the appropriate sympathetic and ordinary nerves) it is assumed, with great probability,

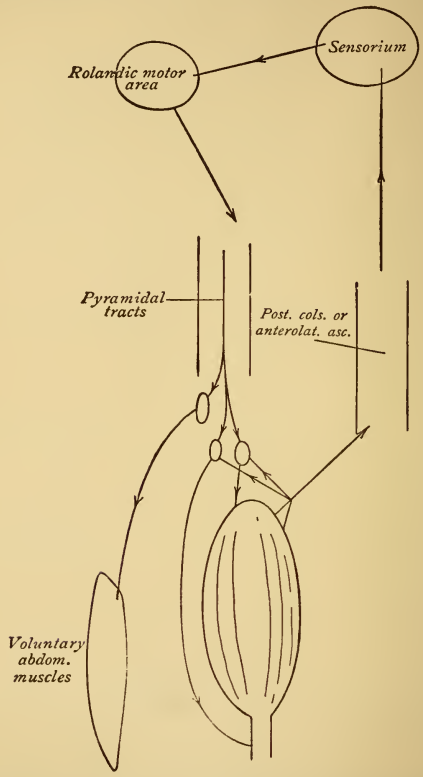

Diagram to represent nerve impulses concerned in normal micturition.

that in the lumbar enlargement of the cord the synapses of the sensory and motor neurons of the lower sacral nerves (probably) are so arranged that afferent impulses, started by the condition or

contents of the viscus, on reaching the cord can, and do in health, diverge in three directions: one impulse or message goes by either the posterior or antero-lateral ascending columns of the cord to the sensorium, and there produces by appropriate synapses a wish to micturate or defæcate; following this message to its termination, and supposing the nervous system is quite intact, the desire can result in restraint of the act or in the act itself, according as circumstances of a social nature are unfavourable or the reverse; the former being one illustration of inhibition (*vide* p. 267). In either case the messages (though of different import) pass down the pyramidal tracts (?) to the appropriate cells of the anterior cornua, and thence by peripheral neurons (1) to the voluntary abdominal muscles, (2) to the sphincter, and (3) to the detrusor musculature of the bladder, influencing them in a manner appropriate to the control or permission of the act. The second and third divisions of the afferent message are, as it were, short circuited, passing simply into the cord by the posterior roots and immediately becoming transmitted by synapses to the efferent neurons controlling the sphincter and detrusor muscles of the bladder, and causing action or inaction of those muscles respectively. It is by this short circuit that micturition and defæcation are provided for in the absence of consciousness. A diagram shows this somewhat complicated description very simply.

Theoretically, then, it is possible for the acts to be interfered with by lesions in any part of this course, but clinically such interferences may be reduced to one of four categories, which, with the possible explanation in each case, may be thus stated:—

1. Retention of urine, distension of the bladder, with overflow and consequent constant dribbling. This is perhaps the commonest form, and is found under many widely different clinical circumstances. Its possibility is due essentially to the fact that the sphincter fibres of the bladder are more powerful than the detrusor fibres. It occurs in—

(*a*) Parturition, especially primiparæ.	Probably then due to a spasm of the sphincter, produced locally by irritative prolonged pressure of fœtus.
(*b*) Operative procedures about perineum, etc.	Probably due to intense reflex stimulus of *unusual* character through *unusual* channels, leading to spasm of sphincter.
(*c*) Tabes dorsalis occasionally.	Probably due to an inability of afferent messages to reach the efferent neurons with sufficient energy.

(*d*) Senile and other mental conditions, especially so-called hysterical.	Probably the original fault lay with highest will centres in checking the voluntary act from deliberate motive; this was followed by blunting of sensorium, while the sphincter still retained its ascendency in physical power.
(*e*) Enlarged prostate and stricture, etc.	Difficulties probably purely mechanical and not nervous at all, except that habit blunts the sensory terminals in the bladder.

2. Complete incontinence, bladder never full, urine constantly dribbling as fast as it reaches the bladder. Indicates essentially a paralysis of the sphincter fibres. This form is rarely found except in—

Acute transverse myelitis of lumbar enlargement.	Due to destruction of the motor efferent neurons, and consequent absolute sphincter paralysis, so that gravity causes the escape of urine.

It is a curious, but very important fact, that such absolute incontinence is never found in purely functional peripheral troubles, and only as the rarest of curiosities even in organic definite peripheral neuritis.

3. A co-ordinated more or less normally complete emptying of the bladder without the intervention of the will or of consciousness. The occurrence of this form essentially indicates that the lumbar (sphincter and detrusor) centres are intact with all their reflex mechanisms, and proves to a demonstration that the higher centres are not essential for the act, but that the lumbar cord is quite competent when necessary to control the entire proceeding. It occurs pretty frequently, *e.g.* in :—

(*a*) Nocturnal incontinence in children (enuresis).	May indicate unusually powerful stimuli (stone, hyperacidity, etc.), or undue irritability of the local nervous mechanism.
(*b*) Transverse myelitis above the lumbar enlargement.	Here it is a localising indication of the position of the lesion, and is of considerable value as indicating that the mischief has not reached the lumbar cord.
(*c*) Tumours and other forms of pressure, causing paraplegia.	Again a localising symptom of some importance.

4. Slighter degrees of interference with co-ordinate micturition.

These form rather a heterogeneous group of cases in their outward manifestations. It includes :—

(*a*) Tabes dorsalis with what is termed stammering bladder; may be present in other forms of sclerosis of cord.	Probably due to irregularity in arrangement and arrival of reflex messages on the afferent side.
(*b*) Temporary retention in spasmodic stricture, stone, etc.	Probably the messages on the afferent side are abnormal in their meaning; possibly also there may be local irritation of sphincter, so that distension with overflow may result.
(*c*) Discharge of urine on coughing, especially in women.	Indicates merely a little weakness of sphincter.
(*d*) Strangury and frequent micturition of cystitis or of enlarged prostate at night.	Probably only indicate the great intensity of *natural* stimuli, the nervous mechanism being intact.

Although we have thus dealt at length with the bladder only, it is extremely probable that defæcation is under precisely similar control, and worked by precisely similar mechanism; but the clinical manifestations of its pathological performance are not so marked, nor so easily followed in detail. This is no doubt due in part to the greater capacity and distensibility of the colon, which acts towards the sphincter ani, like the body of the bladder to its sphincter; but chiefly to the fact that the *bladder* is being *constantly* filled with a *fluid* secretion, while the *rectum* is being *intermittently* filled with a (more or less) *solid* material; a few doses of saline aperient will render the likenesses in action more apparent.

Class C.—Vasomotor reflexes. Of the nerves and paths by which these pass we are profoundly ignorant, except that they seem to run in the so-called sympathetic chain, and to pass to and from the cord with the anterior roots. We recognise the loss of vasomotor control in paralysed limbs and under many other conditions, but we are not able to make much clinical use of our observations as regards localising indications.

From the fact that all reflexes *may* in health or disease be attended with sensory perception of their occurrence, it follows as a legitimate, and indeed inevitable deduction, that the path of the afferent impulse must have at least two dendritic connections in the spinal cord or central system: one with the appropriate motor neuron, and the other with those neurons which ultimately conduct an impression to the sensorium. An anatomical basis has been discovered for this deduction in the division of all afferent nerves into two branches—one going towards motor neurons, and one continuing in the direction of the sensorium.

Seat and Paths of Trophic Influences

But little can be said on this head beyond what has already been noted in the general description of a neuron and the functions of its component parts. It would appear that for the perfect nutrition of all structures they must have a perfect connection with a (at least one, and probably several) neuron cell, the physiological health of which they equally enjoy; failing with it, and recuperating too with it. Obviously the most simple and uncomplicated evidence of this probable connection is most likely to be found and established in the case of the peripheral neurons of the limbs, the dendritic synapses and relationships of which are fairly well understood; and the following clinical examples go far to establishing the general proof of the theory:—

1. The rapid atrophy of the corresponding motor nerves and muscles (or parts of muscles) after complete destruction of all (or some) of the cells of the anterior cornua.

2. The defective growth of all parts of a limb that has been in childhood severely paralysed by anterior poliomyelitis.

3. The changes in the skin and annexa when the sensory nerves or cells on the posterior roots are damaged.

4. The mysteriously rapid onset of bed sores from very slight causes (pressure, warmth, etc.) in many cases of acute myelitis.

5. The equally malignant cystitis and renal trouble that so frequently accelerates or causes death in similar cases of myelitis, notwithstanding most rigorous asepticism in artificial emptying of the bladder.

6. Certain very suggestive dystrophies after experimental division of nerves, *e.g.* the fifth cranial, etc.

Special Sense Tracts

The olfactory, optic, auditory, and glosso-pharyngeal nerves require only to be mentioned here. Their connections will be better studied when dealing with the principles of brain localisation (*vide* Cranial Nerves, p. 303).

These facts of anatomy and associated function form the main foundation upon which are laid the principles of differential diagnosis of nervous diseases; but before proceeding to construct tables from them, there are a few general points and terms to which attention must first be directed.

Inhibition and Interference

By these terms are understood the power which one neuron or group of neurons possesses of so influencing another neuron or group as to check or interfere with in any direction the function which the second mechanism intrinsically possesses. This power may be exercised either voluntarily through the will, of which our daily actions are almost one continuous example, or it may be, and probably is, the inherent function of large numbers of neuron complexes which may thus act without any effect on consciousness, the most constantly occurring clinical example of which is the hypothecated influence of the cerebral and cerebellar cortex on spinal reflexes; the former being supposed to check them, the latter to increase them.

Inhibition and interference constitute almost insuperable difficulties in the way of drawing satisfactory and conclusive deductions from the experimental destruction of limited areas of nerve tissue in cord or brain.

Direct and Indirect Symptoms

By direct symptoms are understood those negative or positive morbid phenomena, directly appreciable by the observer, which arise as the direct result of destruction or irritation of a certain group of fibres with known definite function, *e.g.* blindness from laceration of optic nerve, noises from irritation of the auditory nerve, paralysis from the destruction of the pyramidal tracts, etc.

Indirect symptoms, as the term is used clinically, are really of two kinds: (*a*) those which must be assumed to exist as the plainest corollary from the hypothesis of inhibition and interference, and

may be of a positive or negative character, *e.g.* increased knee jerk from destruction of brain areas; or diminished knee jerk from irritation of similar areas; (*b*) those which might perhaps be more properly spoken of as temporary symptoms, in that they are the result of mere increase of pressure which is often capable of removal; examples of which are very common in tumours of the brain or pons, and hæmorrhage anywhere into the central nervous system or its containing cavity. These pressure symptoms must be allowed to subside in acute (hæmorrhagic probably, or inflammatory) lesions before an exact diagnosis is possible; and in more chronic cases of tumour their possible presence and significance must be very carefully considered.

Inco-ordination

For every movement, whether voluntary or reflex, at least two (and often many more) muscles or groups of muscle fibres are called into action; and in order that the movement may be co-ordinated these several muscles must all act in harmonious relationship to each other (1) in time, (2) in amount, *i.e.* if one of the muscles concerned acts out of its turn, or with a force disproportionate to that exerted by the other muscles concerned, the movement will become disorderly, inco-ordinate, and even perhaps inadequate to its avowed object. Now, by arguments which cannot be introduced here, it is proved that *afferent* impulses from the muscles to the cerebrum and cerebellum (not necessarily, and in fact not usually, causing a conscious perception) are as important for co-ordination as are the motor or efferent impulses to the muscles; and in fact clinical experience almost allows the deduction that if inco-ordination of voluntary movement be present there is a lesion of afferent tracts either of the ordinary class or of the special senses; disseminated sclerosis being almost the only exception, and even here it is assumed rather than proved that the inco-ordination arises from delay in the transmission of motor, and not afferent impulses. It is more commonly complained of in walking (giddiness or stumbling) than in other actions, because this requires very complicated and delicate adjustments of very many muscles, but clinical examination very frequently reveals much that is not complained of by the patient. The following are the principal clinical causes of inco-ordination, and the leading indications pointing to the probable diagnosis.

Cause.	Leading Features.
Peripheral neuritis.	"Pins and needles," and other sensation abnormalities and weakness complained of; skin dystrophy or anæsthetic patches; knee jerks increased or absent; inco-ordination made worse by closing the eyes.
Affections of posterior columns (? of antero-lateral ascending tracts too).	History of lightning pains; knee jerks absent; inco-ordination worse on closing the eyes.
Cerebellar or cerebral disease.	Very possibly severe headaches or optic neuritis if a tumour; if other trouble of chronic nature history clears up the cause; closure of eyes not likely to make matters worse, because inco-ordination is central.
False optical impressions.	A squint, found by appropriate tests; inco-ordination *improved* by closure of the affected eye, or of one if both are affected, because this at once stops the wrong perceptions.
Alcohol or other general action on the sensorium.	History generally obvious; there are also mental alterations, marked in directions other than that of co-ordination of movement; effect of closure of eyes variable.

Reaction of Degeneration

By this term is understood those peculiar changes in the electrical reactions of a *muscle* which are produced in it by the death of the peripheral neurons distributed to it. They are of two kinds—quantitative and qualitative. The essential points to be remembered about R.D. are:—

1. It is a phenomenon of muscle only (nerves simply lose irritability rapidly after severance from their nutritive cells).
2. It always indicates that the peripheral neuron going to the muscle fibre is dead or dying.
3. It is a phenomenon exhibited by each muscle fibril, and consequently is only exhibited in a simple and typical manner when at least a large majority of the individual neurons going to a gross anatomical muscle are affected; thus is explained its common absence in typical form in some cases of progressive muscular atrophy, and other slowly progressive neuron destructions.

4. The quantitative element may be thus represented:—

1st Period.	2nd Period.	3rd Period.	4th Period.
C –	C normal or +	C + +	C – o
F –	F –	F – o	F – o

where C and F represent irritability to the constant and Faradic currents respectively, and period roughly represents a week or ten days or less.

The qualitative element is that—

The natural order of { K.C.C. / A.C.C. / A.O.C. / K.O.C. } becomes { A.C.C. / K.C.C. / K.O.C. / A.O.C. }

during the first two or three periods, but when C and F both become diminished—as between the 3rd and 4th periods—the above change may occur without our being able to make the definite statement that R.D. is present.

Section II.—Diseases of Nervous System

Proceeding now to consider pathological diagnosis, it will conduce to clearer views of analysis if we insert a—

Tabular View of the Causes of Diseases of the Nervous System

1. *Traumatism.*
   - Recent. History obvious.
   - Past. May lead to secondary changes of sclerosing or neoplastic nature.

2. *Vascular Disturbances, with Probable Post-Mortem Eviaence.*

   A. Inflammation.[1]

   (1) Primary of nerve structures, with rapid degeneration and softening. { Cerebritis. / Myelitis of cord. / Neuritis, poliomyelitis, etc. }

[1] The term "inflammation" is used here as the most convenient, because long-established, to express the fact of an acute and very rapid destructive degeneration of neuron cells or processes, or both, which may, however, and often does, occur without very obvious changes in the blood-vessels of the area.

(2) Primary of interstitial or meningeal origin, extending to nerve structures.	Meningitis. Perineuritis.
B. Hæmorrhage.	Vascular degeneration. Aneurysms. Septic arteritis.
C. Plugging of a Vessel.	From cardiac valves, aorta, etc.
(1) Embolic.	From endarteritis obliterans, syphilis, tubercle, etc.
(2) Thrombotic.	

3. *Tumours.*

Malignant, carcinoma and sarcoma.	All primarily irritants, and later destructive by pressure or by invasion of nerve fibres.
Simple connective tissue type.	
Tubercular.	
Syphilitic.	
Abscesses, *i.e.* cysts of inflammatory origin.	
Cysts of non-inflammatory origin.	

4. *Scleroses.*

Primary.	Either of a distinct system, *e.g.* pyramidal tracts, or of quasi-accidental patches.
Secondary.	Following destruction or separation of processes from the neuron cells by any means whatever, *e.g.* lateral sclerosis after cerebral destruction.

5. *Nutritional Disturbances.*—A group of cases more easily comprehended by the imagination than defined in words. It is usually intended to include hysterical and other cases in which recovery is (usually) complete and (not unfrequently) very rapid. They probably all have as their essential foundation a primary lowering of the vitality of a neuron, with loosening of its synapses with other neurons, thus interfering with the transmission of impulses. It is obvious that if this process goes beyond a mere chemical or molecular disturbance the case may soon have to be put in some organic degenerative group.

Any of these pathological changes *may* occur anywhere throughout the nervous system, but each one has its seat of election, and hence, when the locality of a lesion in any case is settled, we are often carried very far in the direction of a diagnosis of its nature,

and *vice versa*. The history of the onset is such an important factor in the diagnosis of the nature of a nerve lesion as to necessitate the insertion of the following table, copied from Sir W. Gowers :—

Disease.	Onset.	Disease.
Acute traumatism.	Sudden, few minutes.	Vascular lesion.
	Acute, few hours or days.	
Pressure and growths.	Sub-acute, one to six weeks.	Inflammation.
	Sub-chronic, six weeks to six months.	Degeneration or scleroses.
	Chronic, over six months.	

An occasional exception may occur, the most important of which is the suddenness with which tumours may now and again cause symptoms either by hæmorrhage taking place into them when otherwise quiescent, or by some slight mechanical alteration in the relation of parts when tumour is present.

Section III.—Differential Diagnosis of Nervous Diseases

With this introductory outline of the structure, functions, and general phenomena of the nervous system, and of its disturbances, we may proceed to the more specialised differential diagnosis of nervous diseases.

The first point in the precise diagnosis of an apparently nervous group of symptoms must be the determination as to whether the symptoms are primarily and essentially caused by an actual lesion (organic or nutritional—Gowers) of the nervous system, or whether they are secondarily, *i.e.* purely reflexly, connected therewith, due to pyrexia, for example, or local disease of other organs.

To exhaust from this point of view in tabular form the differentiation of all the varied phenomena of disease would be confusing and unpractical, if not even impossible, considering that it is only through our nerve structures that we are made conscious of, and adapted to, all the variations of our environment, both local and general, and that the co-ordinate or harmonious working of each and every organ throughout the body is possible.

The following table, then, is but the veriest fragment of an outline of the subject, inserted more for logical completeness of the ideal object of this book than for serious practical utility, though I have endeavoured to make it serviceable as well.

Chronic and Sub-Chronic Symptom Groups

If Caused by Essential Affection of Nervous System.	If Secondarily or Reflexly Nervous.
1. Absence of obvious primary peripheral local disease; especially examine blood and urine and pelvic organs.	1. Such disease is present; *caution*—secondary trophic lesions not to be mistaken for primary.
2. Motor complaints largely in the ascendent; weakness and loss of power in limbs or unsteadiness; movement or its loss rarely associated with pain.	2. Such complaints absent, except with obvious disease of muscles or alteration in outline of joints or limbs: gout, rheumatism, epiphysitis, etc.
3. Sensory complaints often also prominent; usually numbness or paræsthesiæ, if painful most likely of a neuralgic, *i.e.* intermittent and shooting character, or without obvious objective causation.	3. Such complaints practically only of a painful nature, and frequently with obvious causation, *e.g.* chronic pleurisy, cold, abscesses, etc.
4. Complaints of vague alterations from the usual harmonious ease of the acts of micturition and defæcation.	4. Complaints about these acts usually of a straightforward difficulty in act, or definite alteration in frequency, *e.g.* stricture, enlarged prostate, etc.
5. Insidious onset, very variable progression, and unlimited chronicity.	5. Onset fairly marked, progress more uniform and chronicity limited, *e.g.* rheumatoid arthritis, chronic Bright's disease, etc.
6. Reflexes probably altered.	6. Reflexes probably unaltered.

Comments on the Table, and Additional Remarks

1. The examination of blood and urine are expressly mentioned to avoid the mistaking for primary nervous affections the numerous vague and obscure secondary phenomena that occur in anæmia and other primary blood dyscrasiæ, and are the prominent symptoms of chronic and even acute uræmia. A careful examination of the

pelvic organs will often reveal a very substantial cause (neoplasm, parametritis, etc.) for a puzzling sciatica.

2. In children, and even sometimes in adults, it is difficult to distinguish between the refusal to move a limb or part because such movement causes pain (pseudo-paralysis) and the incapability of such movement; the effect of passive movement painful in the former, not in the latter, is the simplest test, and usually sufficient. Other cases in which movement causes exaggeration of already present pain will be referred to in various places where the fact is of use in separating individual complaints.

Inco-ordination of movements, and tremors on effort, are very suggestive of primary nerve lesions, the various forms of Grave's disease being the chief fallacy.

General convulsions and local spasmodic twitchings of muscles constitute evidence of such importance as to require separate consideration.

Vomiting, if reflex from nerve lesions, is usually paroxysmal, very persistent during a paroxysm, and not accompanied by much nausea as a rule; it may be excited by food, but occurs equally independent of it (*vide* p. 158).

Mental changes, except when accompanied by pyrexia, are almost always indicative of an essential nerve lesion, most commonly of the nutritional type when the symptom is primary; when it is secondary to some organic destruction its cause is usually fairly obvious.

Wasting, if general, rarely points to organic disease of the nervous system; but if local, it becomes very suggestive of such a causation (*vide* p. 252).

ACUTE AND SUB-ACUTE SYMPTOM GROUPS

	If Caused by Essential Affection of Nervous System.	If Secondarily or Reflexly Nervous.
Meningeal affections chiefly.	1. Pathological activity, or possibly inactivity of nerves (headache, pain, spasm, delirium, etc.), out of proportion to the degree of fever, and not necessarily abating with defervescence.	This activity or its reverse is more or less proportional to the fever; usually abates with it.
Parenchymatous affections of cords and brain chiefly.	2. Rapid onset of actual loss of function of nerves—paralysis, anæsthesia, etc.	No paralysis, or if so, it is pseudo-paralysis.

On this tabular differential diagnosis of acute symptom complexes it is only necessary to remark that there are but few general points that can be utilised for the purpose. In the cases in which the greatest difficulties and the most serious mistakes are likely to occur the differential points are rather special than general, and consequently the diagnosis will be found in special tables; *vide*—

Meningitis	*v.* Pneumonia, pp. 95 and 96.
Tubercular Meningitis	*v.* Gastritis, pp. 159 and 160.
Infantile Paralysis	*v.* The Exanthemata, p. 292.
Acute Mania	*v.* Delirium Tremens.

Organic *v.* Functional Lesions

We may now continue with general differential diagnosis, on the assumption that the case before us is really and essentially nervous. The next point to determine is whether it be organic or functional.

Even in an elementary work it is impossible to avoid glancing at a definition of these terms. The difficulties in the way of an accurate investigation of the microscopical details of the (whole of the) nervous system have hitherto been so great as to practically prevent a final decision of the question, "Is the picture of structure normal or abnormal, physiological or pathological?" and then of the question, "Did the ill-regulation of function during life depend upon the peculiarities of structure which have been noted?" As modern methods of microscopical technique (hardening, preserving, staining, etc.) become more and more perfect, we are coming more and more to the conclusion that a final answer can be given to the first of these questions, and probably, in consequence, to the second, so that the field of genuine functional diseases is rapidly becoming more and more restricted.

Theoretically, we can conceive that a piece of living machinery that was *originally* (in congenital troubles the same argument applies to the development of structure) capable of carrying out its due function properly and in harmony with the functions of other nervous complexes may work improperly or inharmoniously (= disease) by assuming (1) that its structure has got damaged, or (2) that its supply of nutriment is faulty (excessive, inadequate, or deleterious), or (3) that it individually is suffering from wrong control (imperfect, excessive, or perverted) by other "centres." The last two would correspond to functional, the first to structural,

disease; but it is very easy to see that what was at first purely functional, *qua* a given "centre," may readily result in organic destruction if the untoward influences persist.

As common examples of diseases which are at present frequently thought of as functional, and illustrating the above theory, I would mention—

Epilepsy.—Due primarily to a fine (some day, I believe, to be definitely described) organic change in structure of a centre or centres, kept up or made worse by improper nutriment. Witness the bad influence of some diets (meat) on epileptics.

Hysteria and Neurasthenia.—Due primarily to imperfect nutrition of higher centres, and then a consequent loss of control of these over lower centres which exhibit the symptoms. (Some cases are very close to deliberate malingering.)

Neuralgias.—Often due to organic (inflammatory) changes, but strictly to improper nutrition.

Delirium and other Phenomena of Fevers and of Apyrexial Toxæmia.—Due at once and essentially to deleterious substances supplied to the nerve, often ending in definite structural trouble. (Witness diphtheria, influenza, etc.)

Manias and Permanent Mental Obliquities.—In causation closely allied to the temporary troubles of fevers, etc.; but more frequently the cause ends in fine microscopical changes, which are already being investigated with success.

Occupation Neuroses.—Essentially due to overwork, which in turn leads to, or is associated with, imperfect nutrition by excess of waste products. (These cases are very often ones of definite neuritis.)

Minor Symptoms of Diseases of Brain.—Are in themselves nothing but functional, and are illustrations of perverted control. Typical examples are alteration of reflexes, disturbances of visceral functions, etc.

Considering the very small capability of repair possessed by the nervous system when once a *cell* has been damaged, it would seem almost sufficient to define organic as that which is incapable, functional as that which is capable, of repair. Excepting inflammatory troubles, this is very nearly my meaning in what follows.

Most commonly the point will be obvious from the general aspect and history of the patient, but in cases of doubt the following table will assist us:—

Organic.	Functional.
ONSET.	
If sudden, usually the symptoms are fairly definite and localising.	If sudden, it is less definitely localising, and symptoms more vague, frequently prominently mental.
If chronic, it is still usually fairly constant in character, and consistent in progress.	If chronic, it is very inconstant in features, and inconsistent in progress.
MOTOR SYMPTOMS.	
Paralysis.—Far and away the most common result; if flaccid it is rapidly followed by R.D.; in all cases its distribution is in accordance with known anatomical structure, and objective signs may frequently be found when due to lower level lesions; constant in position when once apparent, at least does not dodge from limb to limb; may be incomplete.	Also tolerably common, but even if flaccid, R.D. never develops; in most cases it is *apparently* due to lower level lesion, and yet no objective signs to be found; its distribution varies from time to time; usually complete in the member complained of.
Spasm.—Rarely due to organic lesion except in Jacksonian epilepsy and the convulsions of meningitis; may occur during sleep; when organic usually a definite known causation in history.	Very common; usually ceases during sleep; no ascertainable cause in the history.
SENSORY SYMPTOMS.	
Not common as the *sole* complaint; if it is the only complaint it is usually definite and localising.	Often enough the *sole* complaint, indefinite and non-localising; definite hemianæsthesia will be unassociated with any features pointing to organic brain trouble, *e.g.* hemiplegia or unconsciousness at onset.
REFLEXES.	
Experimental.—Most usually definitely and permanently altered, and in a constant direction for the individual case.	Variable in different cases which are otherwise similar, and also in the same case from day to day.

Organic.	Functional.
Visceral.—Micturition and defæcation often seriously affected, as also many other visceral functions.	Rarely or never *serious* visceral mischief, but there may be troublesome retention of urine.
Vascular.—Often loss of vasomotor control, limbs cold, and circulation too easily influenced by local conditions; may cause serious anxiety.	Such loss of control only shows itself in turgescent directions (factitious urticaria, *e.g.*), or very temporary coldness and whiteness; never looks serious.
MENTAL CHANGES.	
When present usually persistent, and are associated with other definite indications of organic trouble.	Characteristically variable, and present before illness, or at least not directly caused by it; unassociated with other definitive indications.
TOTAL ASPECT AND COURSE.	
Constant.	Inconstant.
Consistent.	Inconsistent.
Definite.	Indefinite.

The mimicry of organic disease by functional changes is, however, sometimes so complete and so intricate that further details can only be given in special cases.

Assuming that we have determined the case to be one of organic disease, one of the first questions that requires an answer will be—"Is it of the brain or encephalic structures, or is it of the cord (including medulla) or periphery?"

The clearest method of reply is to divide all cases into two categories: (1) acute, a week or less; (2) chronic, a month or over.

A.—ACUTE NERVOUS LESIONS

Before deciding as to an apparently acute case it is exceedingly important to make inquiries into the previous history of the patient, because the following acute incidents in chronic diseases may mislead us:—

Fits in epilepsy.
„ general paralysis of the insane.
„ chronic poisoning (alcohol, lead, etc.).
„ cerebral tumours.

Crises in tabes dorsalis.
Acute features in chronic spinal caries.
" " ear disease, etc.
" " meningitis.

Excluding such cases, the following points are most important:—

	Intracranial.	Cord and some Peripheral Lesions.
ONSET.	Very sudden onset is extremely common.	Such suddenness is rare.
CONSCIOUSNESS.	Very often interfered with in some degree.	Practically never interfered with.
MOTOR SYMPTOMS.		
Cranial Nerves.	Often implicated in some degree.	Rarely or never implicated, unless it be as regards the dilation of pupil, and in post-diphtheritic paralysis.
Paralysis or paresis or excessive movement.	Almost invariably on one side; if on both, nearly sure to be much interference with consciousness or with cranial nerves, as in ventricular hæmorrhage or pontine lesions.	Almost invariably on both sides, owing to nearness of the two sides in the cord, and to the symmetry of peripheral nerves in liability to poisoning; area of disturbance very small if one-sided.
SENSORY SYMPTOMS.	Comparatively rare alone; if present, they are of the hemitype.	Very common indeed, and of the paratype; because motor and sensory tracts are so close together in cord, and both have a nearly equal tendency to suffer in poisoning (?except diphtheria).
REFLEXES.	*Of eye* frequently lost.	Of eye lost sometimes in lesions high up in cord.
	Knee jerk frequently altered on one side	Frequently altered, but more probably on

Intracranial.	Cord and some Peripheral Lesions.
only, unless great excess of intracranial pressure.	both sides, and that without evidence of intracranial pressure.
Bladder and rectum rarely affected, unless consciousness is much interfered with.	Bladder and rectum often interfered with in acute cord lesions, and that without interference with mental clearness.

If the case be a less acute one (over a month), and seems to be stationary or progressing, the following table may be constructed:—

B.—Chronic Nerve Lesions

Intracranial.	*v.*	Extracranial.
History.		
More frequently a history of an acute attack or attacks, and if such be the case, symptoms very possibly stationary or improving; tumours the great exception.		Less frequently such history with stationary symptoms; the symptoms are likely to be progressing, except in cases of definite myelitis.
Motor Symptoms.		
Jacksonian epilepsy (*q.v.*) common enough.		Such uncontrollable movements only take the shape of twitching in legs or arms.
One side most likely alone affected.		Both sides most often affected.
Cranial motor nerves often affected.		Cranial nerves rarely affected except in disseminated sclerosis and in tabes dorsalis, in both of which the lesion may extend to encephalic centres.
Sensory Symptoms.		
Again one-sided and cranial; "pins and needles," and other paræsthesiæ, if present, are of one arm or leg, or one side.		Both sides, and not cranial; "pins and needles," if symmetrical, almost pathognomonic of a peripheral lesion; sensory disturbances in cord troubles nearly sure to be acutely painful or completely anæsthetic.

Intracranial.	*v.*	Extracranial.
REFLEXES.		
Often one-sided change; if both sides, nearly certain to have cranial nerves affected, or mental symptoms.		Equally affected, and that without mental symptoms.
MENTAL CHANGES		
Common.		Never present in a purely cordal lesion, or in one of peripheral nerves.

It is impossible to pursue the plan of general tables for differential diagnosis farther, owing to the mass of details necessary for complete diagnosis, which would be quite irrelevant in many individual diseases. We must now proceed to consider the separate affections, with tables to differentiate those which are more nearly similar.

PERIPHERAL NEURITIS

The existence of lesions of the peripheral nerves, independent of trouble in the cord and brain, has only been determined within quite a recent period; consequently, the first and commonest difficulty in their diagnosis arises from a want of familiarity, not with cases of the disease itself, for they are only too frequent, but with the idea of its existence; and non-acquaintance has, as its natural corollary, non-recognition. It may be laid down as a law that—

"In the gross total of nervous diseases peripheral neuritis, in some one of its many forms, is exceedingly common, and its possible presence must always be borne in mind."

Now, how—in what connections—does it occur, and what are its prominent features? As far as diagnosis is concerned, it occurs in two fairly distinct forms:—

(*a*) More or less localised to individual-named nerves and their branches; a search along which will often reveal the cause of the trouble, *e.g.* a septic wound or tumour causing pressure, though it must not be forgotten that general constitutional poisons, such as rheumatism, gout, or lead, may, and often do, produce such local asymmetrical affections.

(*b*) More universal affection of a large number of nerves (multiple neuritis of authors), almost invariably due to a poison carried by the blood, *e.g.* alcohol, etc., and almost invariably symmetrical.

It is rather a peculiarity of peripheral nerve troubles that they are often either of sensory or of motor nerves only, a point apt to be overlooked; but mixed cases, both sensory and motor, are perhaps more commonly met with. The prominent symptoms are the following:—

1. **Motor Side.**—Weakness and wasting of muscles, with *flaccid* paralysis, and possibly R.D.; certainly R.D. if the case is severe, because the peripheral neuron is the main channel by which nutrition of terminal organs is maintained.

2. **Sensory Side.**—The inflammation acts as an irritant of the peripheral sensory neuron, possibly proceeding even to complete destruction of function; hence we get—

(*a*) "Pins and needles" and numbness. If these are well marked and persistent, and on both sides, they are almost, if not quite, pathognomonic of a peripheral lesion.

(*b*) Pain and hyperæsthesia in the known area of distribution of certain sensory nerves.

(*c*) Anæsthetic patches. These require to be looked for, as the patient has very probably not appreciated their presence. Possibly these anæsthetic regions or areas may be the alleged seat of painful sensations—paradoxical or painful anæsthesia.

3. **Reflex Side.**—Experimental reflexes, especially the knee jerk, either grossly exaggerated (in early stages), or more probably absent; visceral reflexes are almost universally unaffected, so that *serious* disturbance of micturition or defæcation is practically conclusive proof that the cord or brain is affected.

4. **Trophic Side.**—Muscles waste rapidly (*vide* Motor Side); the skin and its appendages show very characteristic appearances; nails become brittle and crack very readily; hair falls out or is temporarily excessive in growth; skin becomes very smooth, shiny, and thin.

5. **Cranial nerves** may be affected, but if so, entirely without general symptoms, and also without any pathological mental phenomena.

If, then, in a given patient some or many of the above are present, we must suspect a peripheral lesion, and inquiry or volunteered statements may then very likely reveal the presence of some antecedent condition known either to predispose or to excite such changes. These, with the leading feature of the resultant neuritis, are given in the following table in rough order of frequency.

Clinical Causes of Peripheral Neuritis

1. Injury, including local inflammation and pressure; septic wounds; necrosis of bone, syphilitic, tubercular, cancerous, aneurysmal, etc., tumours; probably as a sub-group come occupation neuroses (often neuritis).	Causation obtrusive; only the pain or paralysis, or both, make one say neuritis is present; the case is named from the causation, and not necessarily called neuritis.
2. Diphtheria.	Symptoms almost entirely motor, and especially of the eye and palate, but knee jerks almost invariably absent.
3. Rheumatism and gout.	Almost entirely sensory, and pain in the course of a nerve, with a history of gout or rheumatism, the chief guides.
4. Alcohol (with this, but infinitely rarer, must be put poisoning by CS_2, aniline, and a few other trade products).	Both sensory and motor; here "pins and needles" is a very constant feature, with progressive weakness in legs and arms; history of alcohol (possibly denied) or of trade occupation.
5. Lead.	As a peripheral nerve trouble, usually confined to wrist drop, and colic (? nervous); blue line on gums is the guiding feature.
6. Arsenic.	Sensory and motor, usually preceded or accompanied by coryzal symptoms.
7. Diabetes.	Glycosuria; mainly neuralgic symptoms in a given nerve; also absence of knee jerks.

8. Cases occurring in the course of the exanthemata, which present no special diagnostic points; also in anæmia and other cachectic conditions, *e.g.* senility.
9. Malaria, leprosy, beri-beri, and other tropical forms too rare in England to require further notice here.

The presence of one or more of these causes will then often go far towards clinching the diagnosis. Cases will, however, often enough occur in which the cause is not obvious — overlooked, denied, or forgotten—and we may have to depend on the symptoms alone for a diagnosis.

For what affections can the symptoms of peripheral neuritis be mistaken? Simple neuralgia and affections of the cord and brain are the ones chiefly requiring distinction.

Neuritis *v.* Neuralgia

I have adopted the word "neuralgia" here to indicate a pain not due to gross organic changes in a nerve, for the diagnostic problem I wish to try to solve is the following: Is this pain complained of by the patient a purely reflex phenomenon, or at least only due to nutritional (so-called functional) changes, or is it due to gross organic changes, of which actual neuritis is our present example? The following table gives the main points of difference:—

Table

Reflex or Functional, *i.e.* Neuralgia.	Organic, *i.e.* Neuritis.
More likely to be paroxysmal in character.	Less likely to be of this character.
Spontaneous in onset, independent of use of nerve, and, in fact, often better during bodily activity	Pain usually excited by movement, often subsides when a posture of rest adopted, and always worse after use.
Intermittency complete, and history may show long intervals of intermittency.	If intermittent, rarely completely so, and not for long.
Situation often variable from day to day, but almost invariably unilateral in any given attack.	Situation never variable from day to day; more commonly bilateral, but may be either.
If points douleureux present, which they rarely are, pressure on them causes pain at the periphery.	Points douleureux usually present, and pressure on them causes very distinct local pain, as well as perhaps peripheral.
Little or no local tenderness along nerve trunk, and especially a light pressure will cause as much pain as a heavier one.	Generally local tenderness along nerve trunk, and pain much more proportional to amount of pressure.
Wasting or other trophic disturbances never present at first, and only rarely after a long time.	Wasting and trophic disturbances nearly certain to appear rapidly, and may even proceed to R.D. in muscles.

Some of these points can be investigated with a definite result in any stage of the malady, but for others time must be allowed, and often in the early days of symptoms a definite diagnosis is

impossible; but if the cause be organic the nerve is almost sure in a very short time to show very definite signs of loss of function and degeneration, whereas a reflex or functional pain may continue for years without such signs.

To avoid needless repetition, the further distinction between peripheral neuritis and cord troubles can be better discussed after we have considered an outline of the functions and affections of the cord.

DISEASES OF THE SPINAL CORD

Of the real exciting causes of primary affections of the spinal cord we know very little, indeed practically nothing. This is in great contrast to our knowledge of causes of peripheral troubles, but traumatism (external and internal), tumours, acute inflammation, and a slowly destructive process—ultimately ending in degeneration of neurons and a condition known as sclerosis—are the chief clinical causes of those pathological symptoms which we discover by examination. It must be accepted as a fact that the last-mentioned cause (and often also acute inflammation) has, to an extraordinary degree, a habit or power of attacking parts of the spinal cord possessing analogous function, and symmetrically situated on either side of the middle line. So constantly is this the case that these affections are now spoken of as system diseases in opposition to the indiscriminate lesions produced by tumours, hæmorrhages, etc.

System Lesions of Spinal Cord

The systems thus picked out are:—

1. The cells of the anterior cornua;
2. The fibres of the pyramidal tracts (crossed and direct);
3. The fibres of the posterior columns;

and the names given to the diseases thus produced may usefully be tabulated as follows:—

Anterior Cornua—
- Primary
  - Chronic = Progressive muscular atrophy.
  - Acute = Infantile (or adult) spinal paralysis.
- Secondary to acute = Myelitis or tumour pressure, etc.

Lateral Columns or Pyramidal Tracts—
- Primary = Primary lateral sclerosis or spastic paraplegia.

Secondary [1]	= Secondary spastic paraplegia, caused by any destruction of the nutritive cells in the rolandic cortex, or by division or destruction of the main neuron stems from them, such as is left by an acute transverse myelitis.
Posterior Columns—	
Primary	= Tabes dorsalis or locomotor ataxy.
Secondary	= Acute myelitis or tumour pressure, etc.

Besides these pure or simple lesions we have two named combinations—

Combination of anterior cornua and pyramidal tracts.	= Amyotrophic lateral sclerosis.
Combination of pyramidal tracts and posterior columns.	= Ataxic paraplegia.

The diseases in the right-hand column form then the labels of the system lesions of the cord. With the exception of acute anterior poliomyelitis they are all, when primary, *i.e.* without obvious cause, chronic in onset (a year or more). After or during an indiscriminate lesion (tumour, hæmorrhage or softening, traumatism, acute myelitis, all of which except tumour are acute or sub-acute in onset) the symptoms of the system lesion constitute the evidence of location vertically and horizontally. We will now proceed then to tabulate the known functions of the systems to get a firm foundation for differential diagnosis :—

Table

Functions of Systems of Cord

	Pyramidal Tracts.	Anterior Cornua.	Posterior Columns.
Motor	Telegraph wires conveying motor messages from cells of rolandic cortex to dendrites of cells of anterior cornua.	Receive by their dendrites motor messages from pyramidal tracts.	Nil.

[1] This is not usually named as a separate disease, but a symptom-complex or residual condition produced by or left after the lesions mentioned—tumour, hæmorrhage, inflammation, etc.

	Pyramidal Tracts.	Anterior Cornua.	Posterior Columns.
Sensory.	Nil.	Nil.	Sensory telegraph wires conveying messages from skin, muscles, etc., up to medulla and brain.
Reflex.	Transmit impulses from the brain having a restraining influence on spinal reflexes.	Form a link in the reflex chain for any given level.	Also form a link in the chain at a given level.
Trophic.	Nil.	Forms the main source of trophic influences for the peripheral neuron and for the muscles.	Possibly transmit trophic influences to skin.

The Consequent Symptoms of Irritation or Destruction of the Systems are

	Pyramidal Tracts.	Anterior Cornua.	Posterior Columns.
Motor.			
* At the seat of lesion.	Movements or paralysis corresponding to the fibres going out at the level.	Paralysis flaccid, and with R.D. in muscles.	Inco-ordination without loss of power; due to interruption of afferent impulses from muscle by the sensory nerves to the encephalon or head co-ordinating centre; no paralysis or convulsive movements.
* Below the seat of lesion.	Movements or paralysis proportional in extent to the number of fibres destroyed, hence may be merely general weakness; paralysis not flaccid.	No influence on lower cells and muscles.	

* The reason for the difference in symptoms at the seat of lesion, and above and below it, should be most carefully noted. It is that the posterior columns and the pyramidal tracts are merely bundles of telegraph wires, the vast majority of which

	Pyramidal Tracts.	Anterior Cornua.	Posterior Columns.
Sensory.			
* At the seat.	Nil.	Nil	Root pains possibly, girdle sensation.
* Below the seat	Nil.	Nil.	Anæsthesia of opposite side proportional to the number of fibres destroyed; pains possible from irritation of undestroyed fibres.
Reflexes A of Experimental Production.			
* At the seat.	Absent knee jerk in destructive lesions from break in chain.	Absent from break in chain.	Absent from break in chain.
* Below the seat.	Increased from absence of restraint in destructive lesions; in irritative lesions knee jerk is variable.		

are in any given section of the cord merely passing through the seat of section, conducting impulses which actually exhibit themselves lower down or higher up as motion or perception; it is only a very few that at any point leave the cord as the direct continuation of a peripheral neuron with incoming or outgoing message. The reverse is the case with the cells of the anterior cornua, which are all of them in any section individually in relationship with peripheral neurons proceeding from that plane of section (or at least from very closely situated planes); they have no serial functional relationships (of clinical utility at present at any rate) with more distant cells above and below. A similar, I might almost say the same, explanation may be given of the term "root symptoms," an expression frequently used in nervous clinical pathology. It means symptoms, motor or sensory, which can be traced to irritation (usually), or destruction of the peripheral neurons constituting a motor or sensory root of a spinal nerve, either just outside the cord or just within it (after they have left the cells of the anterior cornua in motor cases, before they have been separated in the posterior columns in sensory cases), but leaving the cord *at that plane*; they will obviously be segmental in distribution, *i.e.* a girdle pain or a local pain in the back corresponding to the sensory nerve, or, on the other hand, a twitching or paralysis and atrophy of certain muscles corresponding to named motor nerves.

	Pyramidal Tracts.	Anterior Cornua.	Posterior Columns.
B of Viscera.			
	No voluntary assistance to the act possible, but the centres may work harmoniously.	Loss of expulsive or containing power if in lumbar region, and so dribbling or retention of urine; in regions other than the lumbar result on viscera unknown.	No consciousness of acts, and great mischief may ensue if centres do not work harmoniously.
Trophic to Skin and Muscles.			
* At the seat.	No effect, because peripheral neuron unaffected.	Rapid wasting, with R.D. of muscle fibres or whole muscles, corresponding to destroyed cells.	Bed sores not infrequent, either from anæsthesia or absence of usual trophic influences.
* Below.	No effect.	No effect.	Same as at the seat.

In addition to these three systems we have clinical evidence that the antero-lateral ascending tract of Gowers conveys impressions of pain and touch to the sensorium, and that the gray matter has, amongst other functions, the power of conveying impressions of changes of temperature to the brain, and probably also of controlling the nutrition of the skin and sub-cutaneous structures. Sir W. Gowers has brought forward much evidence in favour of the first proposition; the known thermal anæsthesia of syringomyelia is sugges tive of the correctness of the second; and the third is one possible explanation of the rapid development of bed sores in acute myelitis.

These tables show very distinctly that if a system disease be present in a typical form confusion is impossible, for their symptoms present positive contrasts rather than comparisons. The combined chronic lesions are rather more confusing, and it is moreover true that atypical and indiscriminate lesions are frequent, and hence the differential diagnosis requires some little discussion.

The first caution and golden rule is, "Don't be in a hurry to make a final and definite diagnosis, to find, as it were, a ready-made label

* See footnote, pp. 287-288.

for a case." Consider well *the absence* as well as the presence of positive symptoms, and then let the diagnosis run, "From what I have observed, I have reason to believe that such and such parts of the cord are affected, and, owing to the absence of certain features, other parts are exempt." If the case be typical the appropriate label will then be found, and if atypical no opinion will have been expressed which may afterwards have to be withdrawn.

Indiscriminate Lesions

Inasmuch as an indiscriminate lesion of the cord can only be diagnosed, so far as its symptoms are concerned, by its effects upon those systems or parts of the cord with the functions of which we are more or less acquainted, *i.e.* by the same phenomena—motor, sensory, reflex, and trophic—already several times noted, there follow as immediate and necessary deductions (1) that the history of onset of the illness, and the grouping of the signs, will be of much more importance than their individual presence; (2) that the vertical or horizontal localisation will also assume an equal importance to ascertain if local therapeutical measures offer a prospect of radical cure, *e.g.* tumours.

The lesions themselves are practically only three, viz. traumatism, vascular lesions (blocking, rupture, or inflammatory), and tumours, though syringomyelia and disseminated sclerosis ought also to be enumerated if they had not such special characteristics of their own.

They all of them cause either irritation followed by destruction, or immediate destruction of the cord, and may be classified from that point of view as follows :—

A. Irritative period long, weeks or months, or even years.	Meningitis, simple chronic.
	Tertiary gummata and other tumours of meninges.
	Aneurysmal erosion of bone and compression.
	Spinal caries when products cheesy and possess but feeble potency of irritation, or when gradual bone displacement causes pressure.
	Tumour of the bones.
	Traumatism that has only excited slight inflammatory changes.
B. Irritative period short: hours or days.	Spinal caries with suppuration or sudden displacement of bone.
	Syphilis within two years of infection.

	Tumours of any origin (occasionally).
	Meningitis, acute general.
	Meningeal hæmorrhage.
C. Irritative period almost non-existent.	Acute myelitis (this often occurs in cases where longer irritative period might be looked for, *e.g.* acute meningeal inflammatory affections).
	Hæmorrhage into cord, or softening from blocking of a vessel.
	Traumatism when severe.

Hence it is obvious that it is hardly possible to mistake any of the members of Groups B and C for a system lesion (except perhaps acute infantile paralysis), the history of the onset of the symptoms stands in such absolute contrast. The cases of Group A, too, are almost invariably characterised by one distressing symptom, which is as invariably absent from system lesions, viz. severe local pain or tenderness in the back, a root symptom (*q.v.*, p. 288, footnote).

Besides these two fundamental differences I only propose to give the following brief notes on the diagnosis of indiscriminate lesions.

TRAUMATISM.	A definite history of some violence always to be obtained; in slight cases interference with micturition is the most suspicious circumstance, suggesting serious cord trouble; a chronic meningitis or myelitis may arise from this cause analogous to concussion of brain.
HÆMORRHAGE INTO OR SOFTENING OF CORD.	Exceedingly rare, onset instantaneous, consequent and subsequent symptoms those of an acute myelitis, unless the clot or area of softening be very small, when they will be those of a system lesion, only of acute origin, and probably asymmetrical.
HÆMORRHAGE INTO MENINGES.	Also excessively rare except from traumatism or ruptured aneurysm, in each of which diagnosis is obvious; if a small meningeal hæmorrhage were met with it would be indistinguishable from localised meningitis or tumour except by very sudden onset.
TUMOUR OF MENINGES.	Focal or root symptoms very prominent; complete symptoms more rapid in onset than sclerosis, less so than inflammation; jumpings of a limb with pain in it for some months principal characteristics from pressure on posterior or lateral tracts.

TUMOUR OF SUBSTANCE OF THE CORD.	Very rare; symptoms similar to meningeal tumour, but much more rapidly progress towards a paralytic as opposed to an irritative aspect.

SYSTEM LESIONS *v.* PERIPHERAL NEURITIS

We may now return to peripheral neuritis and those affections of the cord for which it may be mistaken. There are two systems of the cord, disease of which can obviously cause mistakes, viz. the anterior cornua (motor) and the posterior columns (sensory), and that because anterior poliomyelitis and tabes dorsalis are, in fact, lesions of the *same* peripheral neuron as motor and sensory peripheral neuritis respectively.

Pathologically the difference lies in the exact point in the neuron where the trouble begins. This is in the cells of the anterior cornua, and of the posterior roots [1] in the system lesions, with consequent rapid secondary degeneration of neuraxons. While in peripheral neuritis the affection, if not limited to the neuraxon, at least begins in it, and only secondarily involves the neuron cell in severe cases. Clinically we are fortunately able to find a good many differences, so that in the majority of cases but little difficulty arises in diagnosis; though it must be confessed that often also only time and very great patience will but partially unravel the mystery.

A.—THE ONSET

The onset of the system lesions is, as a rule, in marked contrast to that of the peripheral troubles.

Anterior poliomyelitis occurs as:—

1. Acute. (*a*) Idiopathic infantile paralysis.
   (*b*) „ adult „
   (*c*) Part of an acute myelitis.
2. Chronic, or progressive muscular atrophy.

Sclerosis of the posterior columns is, as an idiopathic affection, always chronic.

Now, acute infantile paralysis commences with a sharp outburst of fever, and cannot be diagnosed from a zymotic fever until its special symptom, viz. paralysis, has supervened. Inasmuch as there is a concomitant congestion of the spinal cord, this onset may be associated with a pain in the back, leading to a suspicion of variola or of meningitis; the rash will soon differentiate the former, and the latter will soon be excluded by the subsidence of the pain and the

[1] This is the view now entertained of tabes dorsalis.

very rapid appearance of the paralysis; if meningitis were the trouble the pain would persist and the paralysis (if any) would be long delayed until the inflammation had had time to attack and destroy neuraxons. In adults, while the course of events is precisely the same, the general symptoms are much less pronounced, though the onset is equally rapid; still, the age is in itself very important, for anterior poliomyelitis is as rare in adults as a mysterious, *i.e.* without obvious cause, neuritis is in children, and *vice versa*. On the other hand, peripheral neuritis rarely, if ever, has such an acute pyrexial onset with pain in the back; it may have a smouldering fever lasting several days, or even a week or two (the febrile period in poliomyelitis is at most three or four days, generally only twenty-four or forty-eight hours), but then it has not the pain in the back; if it has the pain in the back (lumbago and sciatica) it will not have the pyrexia unless this be associated with definite joint (rheumatism or gout) trouble.

In those cases in which the acute poliomyelitis is only part of an acute transverse lesion, the early bladder and rectal disturbance and the complete paraplegic anæsthesia will be sufficient to prevent mistakes.

In the more chronic forms of trouble also the onset presents more striking differences than likenesses. Thus, in a peripheral motor lesion there is probably more wasting and paralysis in six weeks to two months than a case of progressive muscular atrophy will show in six months or a year; and, again, the locality will be different, for in the neuritis the whole limb, or whole muscles, will have suffered (except in isolated nerve cases, which will probably have an obvious local cause), whereas in progressive muscular atrophy the changes will be confined to hand or foot (common type), or to shoulder muscles (upper arm type), spreading thence very slowly. The latter disease, progressive muscular atrophy, may be finally dismissed by stating that it has no other nervous features except a progressive weakness going hand in hand *pari passu* with the wasting, and R.D. is not to be obtained typically; while in peripheral neuritis the paralysis is out of all proportion in the early days to the wasting, and R.D. is typically present. The reason for these differences is obvious if we remember that in the system disease the cornual cells suffer one after another in very slow progression, and consequently individual neuraxons with their nutritional functions die and cause atrophy of individual muscle fibres or bundles; while in the peripheral troubles many neuraxons are simultaneously involved.

B.—The Course of Established Symptoms

The system lesions in their course, though sometimes apparently standing still, rarely or never lose their typical physical signs by which they have been diagnosed, but they frequently show improvements or exacerbations quite unaccounted for by anything that can be called treatment or neglect. Peripheral lesions, on the other hand, proceed almost steadily in one of three directions: (1) straight downhill to the grave in less than a year or eighteen months; (2) downhill for so long as the cause acts and a little longer, and then with the cessation of the cause (usually alcohol) steady improvement sets in up to complete recovery, unless and until the cause again operates, when history repeats itself; (3) persist for an indefinite time without any apparent alteration. The perennial root of the tree is attacked in the system lesion; in the other a branch is cut off which may or may not grow again, but, on the other hand, it may be so necessary to nutrition that the whole tree dies.

We will now conclude the subject of cord and peripheral troubles with a few tables of special interest.

Peripheral Neuritis and Tabes Dorsalis

These often have the following in common:—

1. Inco-ordination of movement;
2. Pains in limbs;
3. Absent knee jerks;

but more frequently they differ in the following:—

Peripheral Neuritis.	Tabes Dorsalis.
Inco-ordination seldom a very marked feature; paralysis very common.	Inco-ordination especially marked; paralysis and weakness for simple movements never present.
Pain is in the nerve, never a girdle pain.	Pain shoots along a limb; if a girdle pain present it is very strong evidence (root symptom).
Paræsthesiæ common.	Paræsthesiæ rarely present.
Knee jerks sometimes exaggerated.	Knee jerks never exaggerated.
Viscera never affected.	Visceral crises and disturbance of function common.

Peripheral Neuritis.	Tabes Dorsalis.
Eye affections may be present; if so they are prominently complained of, and may stand as the sole symptoms; optic neuritis or atrophy extremely rare.	Pupillary or oculo-motor troubles nearly constant, but in addition to other symptoms; atrophy of optic nerve frequently found.

Infantile Paralysis will resemble Diphtheritic Peripheral Neuritis in—

(*a*) History of acute illness;
(*b*) Rapid onset of paralysis;
(*c*) Some wasting of muscles;

but the two will probably differ in—

Diphtheria.	Infantile Paralysis.
Acute illness almost certainly with prominent local (throat, vagina, wound, etc.) symptoms.	Acute illness with only general symptoms or some pain in spine.
Paralysis almost always of eye or palate first, and rarely of limbs at all.	Almost always of limbs only, very rarely cranial at all.
Wasting not great, and R.D. typically present only in severe cases.	Wasting considerable, and R.D. nearly constant, in some muscles at any rate.
Very symmetrical paralysis.	Ultimate paralysis characteristically local and asymmetrical.
May get worse for some days after onset; recovery is always practically complete if the patient does not die.	After twenty-four or forty-eight hours any alteration is always in the direction of improvement; recovery is never *absolutely* complete.

Ataxic Paraplegia *v.* Peripheral Neuritis

The combined motor and sensory phenomena may arouse suspicion of some peripheral affection, but the course of the cord disease is in itself almost sufficient to separate it from the latter; the cord trouble is always slow in onset, and months elapse before it advances far, while the neuritis is almost invariably acute or subacute. The very marked inco-ordination, too, of the cord trouble, especially taken with the exaggerated knee jerk, adds further confirmatory differentiation.

Amyotrophic Lateral Sclerosis *v.* Peripheral Neuritis

Dr. Gowers says, "The wasting from disease of single nerves or at a plexus is sufficiently distinguished by its limitation, coupled with its rapid onset and associated sensory symptoms"; and from some forms of multiple neuritis, he says, "It is necessary to wait for slower wasting in other parts than those first affected before a diagnosis can be made; a careful search will generally reveal other symptoms of neuritis, and a known cause is usually obtrusive."

Lateral Sclerosis *v.* Peripheral Neuritis

Any affection of the pyramidal tracts can only be mistaken for a case of pure motor peripheral trouble. If sensory phenomena are present to an extent sufficient to attract attention the disease is not primary lateral sclerosis. The cases of motor neuritis which could rouse an idea of lateral sclerosis must be either of arms or legs. Now, if it be of the arms, the note at the bottom of p. 287 shows us that if the lateral columns are affected as high as the origin of the brachial plexus, the knee jerk is exaggerated and ankle clonus probably marked; but, *per contra*, the peripheral brachial nerves have no such influence on the rest of the cord, and there will be no disturbance of reflexes lower down. The same test will also apply if the trouble be in the legs, for, except in the earliest stage of neuritis (and that arising in a manner and under circumstances which preclude all idea of lateral sclerosis), the knee jerk is absent and ankle clonus not to be obtained.

Peripheral Neuritis *v.* Disseminated Sclerosis

Insular sclerosis most usually offers a fairly typical symptom complex which cannot be mistaken for anything except functional (or hysterical) nervous disease; but inasmuch as its very name implies a random distribution, it is possible that cases may arise in which the peripheral nerves are suspected.

Of the many symptoms that may occur in disseminated sclerosis there are three which, notwithstanding its indiscriminate distribution, are almost constant, viz.—

1. Intention tremors;
2. Nystagmus;
3. Increased knee jerk;

and it is as rare to find a case of peripheral neuritis exhibiting these

three in combination without other obvious indications as it is to find a case of disseminated sclerosis in which they are not all three present.

Disseminated Sclerosis *v.* Hysteria or Functional Troubles

To distinguish the early stages of sclerosis from what is usually accepted as functional disease is probably to try and make a distinction between identicals, for the death of neurons by sclerosis is in all probability preceded by a stage of functional debility in which recovery is certainly possible, but as the recognition and demonstration of this recoverable condition is all-important from a therapeutical and prognostic point of view, I have drawn up the following table of points most worthy of attention in rough order of importance.

Functional.	Established Disseminated Sclerosis.
1. Tremors very common, but not so violent as to prevent completion of intention.	1. Tremors so violent as frequently to quite frustrate intention.
2. Nystagmus very rare and ill-marked; double vision rare except very temporary.	2. Nystagmus common and very distinct; double vision not uncommon.
3. Knee jerks probably glib, but variable from time to time.	3. Knee jerks grossly exaggerated and invariable; should a patch have caught the reflex chain they will be absent, but still invariable.
4. Speech probably voluble, or obstinate silence; seldom scanning, varies from time to time.	4. Speech scanning usually, and not variable.
5. If bladder trouble at all it is retention, and pain not complained of.	5. Bladder trouble frequent; incontinence, stammering bladder, and even painful retention.
6. Optic neuritis or atrophy unknown.	6. Optic atrophy if present is conclusive, for it *is* sclerosis.
7. Recovery rather sudden and complete, and possibly no return; if return it is sudden and in the same guise.	7. Apparent recovery curiously frequent, but return of symptoms soon occurs; may be in another guise.
8. Sensory hemi-anæsthesia not uncommon with recovery.	8. Hemi-anæsthesia rare, and what anæsthesia occurs is little likely ever to be removed.

Functional.	Established Disseminated Sclerosis.
9. Perversion of will power a frequent cause, or concomitant, and very variable at times.	9. Perversion of will power not often observed to any extent; if it is present it remains.
10. Spasticity and inco-ordination variable from time to time.	10. If spasticity or inco-ordination occurs it is likely to be permanent.
11. If any wasting occurs it is likely to be general, and very slow in onset, and arises from mere disuse.	11. If wasting occurs it is likely to be local and fairly rapid, though when lateral columns are affected this will not hold.

AFFECTIONS OF MEDULLA, PONS, AND CEREBELLAR PEDUNCLES

In dealing with the peripheral nerves and spinal cord I have dealt at some little length with the points of differential diagnosis because the cases are more commonly met with, and because the problems of diagnosis are comparatively simple in themselves if due attention is paid to the elements that lead to conclusions.

In dealing with intracranial troubles we are introduced at once to so much that is absolutely unknown, and still more that is only guessed at or in dispute, as regards anatomy and physiology, that complete diagnosis is frequently impossible, and I shall only attempt to sketch a clear outline of the principles of regional and pathological diagnosis, and introduce independently a few important subjects which are but ill remembered and worse understood by students.

An accurate knowledge of the precise regional relations of the various tracts of fibres and groups of cells whose functions are more or less known is absolutely essential for a clear understanding of the diagnostic problems offered by disease in this region. The best way to learn this is by careful study of sections compared with the figures to be found in every text-book of anatomy. They may be tabulated for study thus :—

Fibres of Pons, Medulla, and Cerebellar Peduncles

A.—*Old Tracts already noticed in the Cord*

1. Pyramidal, crossed and direct;	Constitute the crossing of the pyramids, and then the pyramidal tracts through the medulla, pons,

functions purely motor.	and cerebral "crura." In tracing these *down* from the motor cortex it must be noted that some of them turn out to join the nuclei of origin of the motor cranial nerves, in which case the relationship between cortex and nucleus is always *crossed*, precisely the same as that of cortex and anterior cornua of cord.
2. Antero-lateral ascending tract; function to convey centripetal impulses.	Some are traceable as fibres in the formatio reticularis, reaching thus the superior cerebellar peduncles and so the cerebellar cortex; others are lost in the formatio reticularis, and their further course unknown.
3. Direct cerebellar tract; function centripetal (? entirely).	Form a large part of the restiform bodies or inferior peduncles of cerebellum. This is the only known ending of these fibres.
4. Posterior columns: function centripetal.	End in synapses with the cells of the funiculi gracilis, cuneatus, and rolandi.
5. Bulk of unnamed white fibres of cord, function nearly or quite unknown unless commissural.	Constitute probably the bulk of the fibres of the formatio reticularis. Their destination is unknown and also their function, though they are assumed to unite successive segments of the mid-brain.

B.—*New Tracts of Fibres, or in some cases New Names for Old Tracts*

Originate in.	Tract.	Reaching to.
1. (*a*) Cells of nuclei gracilis, cuneatus, and rolandi.	Of formatio reticularis.	(*a*) Function afferent, reaching the inferior peduncles of cerebellum as internal arcuate fibres, passing to cerebellar cortex.
(*b*) Unnamed white fibres of cord.		(*b*) Reach cortex of cerebrum and cerebellum, or end in cells of formatio reticularis (*vide* 5 above).
(*c*) Olivary body and other cells in the region of the upper medulla.		(*c*) Many go as arcuate fibres to cerebellum, many also of unknown destination and function.
2. Direct cellebellar tract of cord, internal and external arcuate fibres.	Of inferior cerebellar peduncles.	Cerebellar cortex and nuclei.

Originate in.	Tract.	Reaching to.
3. Cells of nuclei gracilis, cuneatus, and rolandi, also independent cells in formatio reticularis, and many of cells of the olivary body.	Arcuate fibres.	Functions probably afferent, reaching cerebellar cortex by inferior peduncles.
4. Begins by axons from cells of nuclei gracilis, cuneatus, and rolandi, added to by axons from cells of formatio reticularis, of olivary body, etc.	Fillet.	Is the principal afferent (? entirely) passage to brain from the cells in medulla, reaching to corpora quadrigemina and optic thalamus.
5. Cells of reticular formation at one end, cells near the nucleus of third nerve at the other, and from intermediate cells.	Posterior longitudinal bundle.	Probably motor in function, serving for connections between motor cranial nerves, especially in their associated (quasi-reflex) movements, *e.g.* lips and tongue, the two eyes, etc.
6. From cortex of one cerebellar hemisphere.	Transverse fibres of pons.	To the other hemisphere, probably engaged in co-ordination.

Cells of the region

Of funiculi— Gracilis, Cuneatus, Rolandi,	Form some of the neuraxons of— (*a*) Fillet. (*b*) Internal arcuate fibres. (*c*) Others of unknown destination.
In formatio— Reticularis.	Form neuraxons going to— (*a*) Reinforce fillet. (*b*) Connect different segments. (*c*) External arcuate fibres. (*d*) Reinforce posterior longitudinal bundle.
Cells of cranial nerves from three to twelve.	Form the neuraxons of their respective nerves.

Now, in applying these facts of anatomy and physiology to the problems of diagnosis, it cannot be too strongly insisted upon that in intracranial lesions, just as in peripheral (and cord) ones, there is

nothing essentially mysterious or beyond the power of the youngest student to grasp. The mystery, so far as there is any, lies simply and solely in our ignorance of the exact path of nerve impulses, and of the amount and nature of control and interference exercised by one neuron group over another. Lesions, of whatever nature, affect the structure and function of individual neurons in precisely the same way here as elsewhere, and this affection (whether of defect or excess) it is that constitutes our means of localising diagnosis.

The elementary facts to grasp are :—

1. That every anatomical nerve in the body has the power of communicating with, or of receiving messages from, the cortex of cerebrum and cerebellum for purposes of harmonious functioning in the healthy body.

2. That for some reason (several explanations of the fact have been offered) that portion of the cerebral cortex which is thus in connection with a peripheral nerve is in the opposite named (right or left) hemisphere to that side in which occurs the peripheral distribution of the nerve in question.

3. That, *per contra*, the relationship of the cerebellar cortex to a nerve is an uncrossed one.

4. That this crossing of impulses and of fibres lies—

A. For motor nerves and impulses—

(*a*) In the lower part of the medulla, so far as spinal nerves are in question.

(*b*) For the cranial nerves, just a little nearer to the cortex than the nuclei of origin.

B. For sensory nerves and impulses—

(*a*) Distributed throughout the cord and medulla for the spinal ones, each nerve crossing at once.

(*b*) For the cranial nerves, just a little nearer the cortex than the nuclei of origin.

5. It is at once an obvious and necessary deduction from these elements of crossed influence that a lesion of a strand of fibres above, *i.e.* nearer the cortex cerebri than the nucleus of origin of the peripheral nerve in question, must exhibit its effects on the opposite side of the body to that on which the lesion lies. If it be at, or on the peripheral side of, the nucleus, its effects must be exhibited on the same side as that of the lesion; and further, from what has

been said on p. 252 about the function of the neuron cell, must almost inevitably be associated with some trophic alteration (excess or defect) in the muscles affected.

6. A self-obvious statement, but one too frequently forgotten, that the closer the nerve fibres (or cells) lie together the more likely are they to be involved together by a small lesion. In this fact, however elementary it may seem, lies the clue to at least one-half of the localising problem. Thus, taking the motor side and a destructive lesion as an illustrative example, a cortical or subcortical small lesion may easily be understood to be capable of producing a monoplegia, but the nearer we get to the (anterior two-thirds of the posterior limb of the) internal capsule, the more fibres will be caught by the lesion, and the wider will be the peripheral area of disturbance, until when we arrive at the crura cerebri, pons, and medulla the motor fibres are collected so closely together that it is almost impossible but that a paralysis shall be at least a hemiplegia, and possibly even a complete paralysis of all four limbs. Further, speaking of our present region—the mid-brain—it is almost impossible but that some at least of the motor cranial nerves shall have participated in the disturbance; for this reason they give us tolerably exact information of the locality of the trouble. This will at once explain the following clinical experience, viz. that *given a hemiplegia*, then if it is due to a lesion—

In Crus Cerebri.—The third nerve on the opposite side is nearly sure to be paralysed. Any other cranial paralysis will be on the same side as the paralysis of the body.

In Upper Pons.—We may look for some weakness in the opposite fifth nerve (motor part), and possibly some anæsthesia, though there is a considerable distance between the motor pyramidal fibres and the sensory root of the fifth.

In Lower Pons.—Very probably a paralysis of the opposite seventh, because its peripheral course lies so close to the motor fibres which are destroyed. (The explanation of the contraction of the pupils, a constant feature, is not quite so obvious.)

In Medulla.—We may look out for a paralysis of the opposite sixth, very possibly of the opposite seventh, because the seventh nerve winds round the sixth nucleus. (A paralysis of both sixth nerves is not such a precise indication, owing

to their exposure to pressure between the pons and the bone.) In the lower part of the medulla one twelfth nerve or a spinal accessory is very likely to be affected, leading to deviation of protruded tongue, or to paralysis of a vocal cord.

CRANIAL NERVES

The last paragraph has shown the importance of these nerves as indicators of locality of a lesion. I propose now to add a few notes on the individual nerves and their lesions.

First, or Olfactory.—This, strictly speaking, is not a nerve but an integral part of the cerebral cortex. Clinically, if the sense of smell is impaired, we must first suspect and investigate the external cavities. If these are declared free from suspicion the trouble may be situated in the anterior fossa of the skull, causing pressure on the olfactory bulbs. The cortical connections of the bulbs are supposed to exist in the tip of the temporo-sphenoidal lobe, so that in cases of suspected abscess or tumour, interference with the faculty of smell is a suggestion that the lesion is not far from that situation.

Second, or Optic.—Like the olfactory, is a distinct outgrowth of the brain, with a primary connection with the corpora quadrigemina. Its cortical representation is found (*a*) in the occipital lobes for coarse optical impressions, *i.e.* for an appreciation of the fact that something is affecting the optic nerves; (*b*) in the supramarginal and angular gyri round the end of the sylvian fissure for optical perceptions and judgments, *i.e.* for an appreciation of the precise object that is stimulating the optic nerves, and its exact nature and associations. Hence, as regards localising deductions from the nerve, we have—

(*a*) Optic neuritis (*vide* p. 324).

(*b*) If hemianopia be due to a lesion of the optic tracts the pupil will not react when light is thrown on the blind half of the retina, because the lesion is in front of the centre for reflex of light; if it lies between the corpora quadrigemina and the optic lobes the pupil will react, but the patient will not perceive that anything is stimulating the retina; if the lesion is between the occipital and the supramarginal lobes he will perceive the object,

but will not judge its nature correctly. (This is the condition known as word blindness when testing a case of aphasia.)

Third, Fourth, and Sixth. — These nerves, taken collectively, form the motor apparatus (with muscles) of the eye-balls, built and arranged to subserve the purpose of single vision with two eyes, for which very accurate associated action of the muscles is required. This is provided for undoubtedly by the close approximation of the nuclei of the nerves, and by the posterior longitudinal bundle serving as a rapid means of communication; hence it certainly follows that nuclear lesions of these nerves or of the posterior longitudinal bundle will cause squinting, or at least double vision (squint is merely a deviation of the visual axes from their normal position visible to the observer). Such lesions are known, though not very common; their exact discussion would be too intricate to follow out here. It must not be forgotten that these three nerves lie very close to one another and to the orbital branch of the fifth at the entrance to the orbit, so that a tumour or sclerosing lesion in that situation may produce a total paralysis of all four nerves.

Simpler problems are presented by considering the individual nerves and their branches, *i.e.* peripheral lesions.

Third Nerve.—If affected by a crural lesion it is probable that there will be a complete paralysis of the whole of the third on the opposite side to that on which the paralysis of the body is said to be. If affected in the more peripheral parts of its course, it is more probable that the paralysis will be incomplete, *i.e.* of one or more branches only. The reason for this is better discussed along with pathological diagnosis.

Fourth.—To this nerve the same remarks apply as to the third, remembering that it only supplies one muscle, viz. the *superior* oblique, which assists in *downward* movements of the globe. Hence double vision on looking downwards only.

Fifth.—When affected at or above its nucleus never, according to Bastian, exhibits trophic, vasomotor, or secretory disturbances of function; when affected in its peripheral course does exhibit such disturbances in the appropriate area of distribution.

Sixth.—As above remarked, the exposed position of these nerves between the pons behind and the bone in front deprives a double paralysis of much localising value, but if one only is affected we have strong ground, *in an otherwise doubtful case*, for believing that the

lesion is in the medulla or in front of the pons; in front, that is, as regards the cranial fossæ. If one sixth and the same named (right or left) seventh are involved together, we can fix the lesion pretty accurately as at that spot in the medulla where the seventh nerve winds round the sixth nucleus.

Seventh.—In small lesions at or above, *i.e.* nearer the cortex, its nucleus, it is a clinical fact that the occipito-frontalis muscle almost invariably escapes, probably because the muscle is most commonly used reflexly, and stimuli reach it by the posterior longitudinal bundle. If the nerve is affected between the brain and the bottom of the internal auditory meatus it is very likely that deafness of the same ear may be found, because the seventh nerve lies in actual contact with the eighth. In lesions beyond this point, up to the exit from the stylomastoid foramen, it is likely that the paralysis will be absolutely complete. Beyond this point individual branches may be caught by a lesion, but we shall have probably gross manifestations of a swelling (parotitis, etc.) or inflammation to help us outside the skull. It is also to be noted that in peripheral lesions there will be no associated hemiplegia.

Eighth.—In central lesions it is not likely that the auditory nerve will suffer alone, its nuclei are too wide spread. In peripheral lesions it will be likely to suffer alone, or, as above noted, in conjunction with the seventh only of the same side.

Ninth, Tenth, and Eleventh.—Individual lesions of any of these nerves alone at their nuclei are excessively rare. A central lesion of any of them will, therefore, locate itself in the medulla by being associated with an affection of all three. A peripheral lesion will define itself by features external to the skull.

Twelfth.—The tongue is so intimately connected with the power of articulate speech as to lead to a conclusion that the cortical representation of the twelfth nerve is to be found in Broca's convolution on the left side, and in a corresponding area on the right hemisphere; and we find that a lesion on the left side (? on the right side in left-handed people) in Broca's convolution, or between it and the twelfth nuclei, disarranges the *function* of the tongue so far as that subserves speech (pure motor aphasia).

Again, in deglutition, and in practically all uses of the tongue, the two halves are so accustomed to work together that it would seem a great convenience (or, probably, almost a necessity) that they should be capable of being stimulated from either side of the

brain. Furthermore, as a matter of anatomical fact, the nuclei of the right and left twelfth nerves lie so close together in the floor of the lower half of the fourth ventricle as to be almost intermixed. Hence, as a matter of clinical experience, we find that (barring speech defects) in lesions above the nuclei, if a one-sided paralysis of the tongue occurs, it is likely to be incomplete and also to be comparatively temporary, and certainly unassociated with any trophic lesions. In lesions at the nuclei the paralysis is almost sure to be bilateral, and (*vide* p. 252) associated with atrophy. From lesions peripheral to the nucleus we may again get a one-sided paralysis, but this time with trophic lesions.

DISEASES OF THE BRAIN

What we *know* about the brain is, that it is composed of neurons and supporting (mechanical and nutritional) tissue. The former so arranged and connected as to subserve in the best possible manner the great purpose of bringing us into more or less harmonious communication with the world at large—co-ordination of functions in the very widest meaning of the word. Here resides the consciousness of change in the environment, both internal, of organs, and external, of the body, and here is initiated the necessary process to adapt everything to that change.

What we *hypothecate* is, that through these neurons, and their synapses with one another, messages can with very great ease pass from one hemisphere to the other (commissural fibres), or from every part to every part of the hemisphere of the same side (internuncial fibres), and also with both direct and crossed influence from cerebral to cerebellar cortex and mechanisms.

To completely analyse the tracks by which this is done—even those which are tolerably well known—would require a large volume, and would in the present work serve no useful purpose. So far as bedside diagnosis is concerned, the principal difficulties in the way of accuracy are: (1) want of precise knowledge of actual structural continuity of tract with tract; (2) a still greater lack of precision in knowledge of functional continuity; (3) the fact that direct pathological lesions, and still less their effects as regards pressure, so very seldom confine themselves to those individual tracts, or tracks, about which we think we know something.

The following tables give some of the principal points of localisation, either approximately proved or strongly suggested:—

CORTEX OF THE BRAIN—	
Ascending parietal, ascending frontal, and corresponding median cortex.	Possess the function of initiating the *motor side of movements* in various parts of the body on the opposite side. In this area of cortex are represented in order from above downwards the foot, leg, arm, face, so that a pure cortical destructive lesion produces weakening of these movements. For irritative lesions, *vide* Jacksonian Epilepsy, p. 309.
Frontal.	Suggestively the seat of connected thought and reasoning; lesions here have, in some cases at least, impaired this power, *e.g.* general paralysis of the insane. Indubitably this region comes into structural continuity with the anterior limb of the internal capsule, and so functionally is supposed to be connected with the cerebellar and occipital cortex.
Occipital.	Probably in the main the seat of subjective consciousness of sensory stimuli; it is almost certainly proved also to be the seat of the primary registration of optic impressions.
Supramarginal and angular.	Proved almost conclusively to be the seat of the final registration of optic impressions, *i.e.* visual concepts as opposed to mere percepts, and to be for this purpose in close connection with speech (on its motor side) and appropriate common motor areas. These facts are such, at least, for the left hemisphere (*vide* Optic Nerve).
Superior temporo-sphenoidal.	Stands in precisely the same relation to auditory stimuli that the supramarginal does to visual ones (*vide* Auditory).
Tip of the temporo-sphenoidal lobe.	Represents the olfactory sense in a similar though perhaps less efficient manner.
The rest of the cortex.	Very few or no facts essentially proving a local or definite function.
CORTEX OF CEREBELLUM.	All we can say about this is, that when, by damage to its peduncles, messages

are unable to reach the cortex, defects of equilibration become a marked feature in the disease, but we cannot say more precisely how or why these are caused.

White Matter underlying Cortex—

Inasmuch as this is engaged in carrying messages to and from the cortex, it is difficult, or even in many cases impossible, to differentiate lesions of it from those of the overlying gray matter. All that can be said, in general terms, is, that the nearer to the cortex the more likely is subjective consciousness—apart from a mere unconsciousness in the ordinary sense of the word—to be implicated in the pathological disturbance, and on the motor side the less likely are we to get mere twitching (or loss of power) in groups of muscles as opposed to actual purposive (but unconscious) movements or weakening of such movements. We know a little about some tracks, or tracts, in the white matter. Thus:—

That in the frontal lobes is collected mainly into the **anterior limb of the internal capsule.** We do not know much about lesions of this, except that we should expect inco-ordination of movement to some extent, and incoherence of thought, *i.e.* we only know it so far as we know the function of the frontal cortex.

That corresponding to the motor cortex is gathered together as the **corona radiata,** and then more concentrated still in the anterior two-thirds of the posterior limb of the internal capsule. As it conveys the motor impulses lesions of it will cause hemiplegia or hemiconvulsions.

That corresponding to the occipital cortex conveys impressions to the sensorium from everywhere, **per the posterior third of the posterior limb of the internal capsule,** and lesions should produce hemianæsthesia and lesions of sight corresponding to what has already been said on that subject: also interference with the afferent impulses necessary for co-ordination.

That of **corpus callosum** can only be said to connect the two hemispheres; of its special function, and of evidence of its lesions, we can say nothing here.

Basal Ganglia with their Fibres and Cells—

Of the corpus striatum, optic thalamus, and cells of the tegmentum of the crura cerebri, it is impossible to say anything definite without going into minute detail. We recognise in them anatomically complete neuron groups (cells and processes) in very

intimate connection with one another and with cortical groups —evidently very complex and delicate mechanisms for the rapid distribution and adjustment of nerve stimuli; but, notwithstanding the attention they have received of late, we are still almost as much in the dark now as ever we were as to the *precise* part played by them in conscious, or in reflex life. We can still only say that the corpus striatum probably has something to say to the correct adaptation of means to ends on the motor side, while the optic thalamus and tegmentum play a similar rôle in connection with the correct appreciation of afferent stimuli on their way to the cortex. Of the corpora quadrigemina and gray matter in connection with them we can say that they play a conspicuous part in the correct manipulation of the eyes, including in that term not only gross movements but also accommodation and reflex to light.

We may now conclude this elementary outline of localisation of the brain functions by a note on one or two points of special diagnostic interest as illustrations.

ON THE MOTOR SIDE

A.—Jacksonian Epilepsy—

This term has been given—in honour of the first accurate investigator of the phenomena—to attacks of convulsive movements which commence locally in an extremity, or in the face, and are due to an irritant lesion of some kind in that portion of the rolandic (or motor) area corresponding to the given peripheral commencement of the movements. Any given attack may be quite local, and limited in distribution almost to the spot originally attacked, or it may be spread more and more widely so as to involve the other limbs of the same side, and even those of the other half of the body, and may even end in unconsciousness, hence it may closely resemble idiopathic epilepsy. The important points to remember about it are, that for the *same* patient—

(*a*) It invariably begins in the same place (thumb, great toe, and mouth are the commonest).

(*b*) It invariably spreads in the same direction, though not necessarily to the same extent; the extent apparently depends upon the nature and amount of the *temporary* exciting cause of extra activity, but the direction is probably governed by lines of least resistance, which soon become established in any patient, and remain constant for that patient.

(*c*) It is always followed by an (at least temporary) paralysis of the convulsed muscles which—

(*d*) Is always most marked in the muscles first affected.

Thus its presence is a very strong piece of localising evidence for an irritant in a particular part of the rolandic region. In the above particulars it may closely resemble idiopathic epilepsy, especially if the aura of the latter is local, but the following points will usually serve to distinguish them :—

Jacksonian.	Idiopathic.
1. In most cases either a marked and obvious cause, *e.g.* traumatism, or evidence suggestive of a neoplasm as cause.	If cause be present it will be in the shape of a family history or something general, not local. Absence of evidence of gross intracranial disease other than that afforded by the fit.
2. The commencement always local, and movement precedes sensation.	Onset not often local in the situations chosen by Jacksonian ; when it is thus local sensation precedes movement.
3. No tonic stage, at least not marked.	Tonic stage nearly constantly present.
4. Comparatively rarely goes so far as general loss of consciousness.	Loss of consciousness is a constant feature, though it may be only momentary.
5. After a fit, paralysis, or at least weakness, is a marked feature in the locality of the commencement.	Paralysis after a fit not present, a feeling of tiredness in general the only evidence of motor distress.
6. No sleepiness after a fit.	Sleepiness very frequent after a fit.

B.—Other Motor Disturbances of Voluntary Movements—

Besides Jacksonian epilepsy, the following involuntary movements are usually assumed to be due to damage of the brain, because (1) they are usually associated with mental deterioration ; (2) they cease during sleep as a rule ; (3) they are more complex in gross aspect than we are accustomed to see in pure spinal movements.

Paralysis agitans. Coarse rhythmical movements, usually of the head and hands, more marked during inaction ; often, too, these are of the hemi- type, another argument for a brain origin.

Chorea.	Coarse, involuntary imitations of purposive movements, continuous during waking hours; also often of hemi- type.
General paralysis of insane.	Principally of lips and tongue during speech, also seen in the limbs; the morbid anatomy of this disease is known to be a fine cortical sclerosis.
Chronic alcoholism or mercurialism.	Coarse tremor of hands upon attempted fine movements.
Athetosis.	Peculiar rolling movement of affected digits; frequently post-hemiplegic, and then indicates that the lesion is probably near the cortex.

C.—Symmetrically used Muscles—

In those symmetrical muscles of the two sides of the trunk and face which are almost never used independently in health, *e.g.* intercostals, eye muscles, etc., complete paralysis is never seen from a one-sided central lesion; the explanation of this, known as Broadbent's hypothesis, is that such muscles are stimulated into action with nearly equal facility from either hemisphere, either through the corpus callosum or through mechanisms in the mid-brain (? cerebellum).

ON THE SENSORY SIDE

Information and facts are much less definite and numerous in considering afferent than efferent impulses. When once the sensory impulse has reached the medulla on its centripetal journey we know little or nothing about it and its neuron complexes, or series of complexes, till it reaches the posterior third of the posterior limb of the internal capsule. It is clinical observation that has taught us that afferent impulses are absolutely necessary for the co-ordinate and harmonious working of the whole body; but, so far as incoordination in the narrower sense of mere movement is concerned, we have only one fact of importance to guide us, viz. that if incoordination be due to spinal or peripheral lesions, as in tabes, in all probability the knee jerk will be absent, if it be due to intracranial lesions the knee jerk will be either natural or exaggerated.

Hemianæsthesia—

Due to an organic cause is an excessively rare phenomenon *when standing alone*; when with hemiplegia it almost conclusively proves a lesion of the internal capsule, because it is only here that

the sensory fibres are sufficiently close together and to the motor ones to be thus affected by a lesion small enough to not cause such an interference as will either rapidly prove fatal or have such an effect upon consciousness as to prevent trustworthy observations.

N.B.—This last statement should be carefully borne in mind, for it is the clue to the reason why many bizarre effects in cerebral troubles have not been noted; if the lesion is great enough to damage tracts widely separated, it is either multiple or so large as to cause rapid death.

Hemianæsthesia *as the sole evidence of disease* is almost invariably due to functional and recoverable causes probably affecting the internal capsule.

ON THE MENTAL SIDE

If there be no pyrexia or other evidence of local disease outside the nervous system, then, *qua* localisation from mental phenomena, all we can say is that the disease is above the pons and crura, and in all probability is in the cortex of the cerebrum. They arise in all probability from a loosening of, or at least alteration in, the synapsial connections of the cortical neurons. Clinical experience would tend to suggest that the frontal lobes have more to do with the mind than any other part, but nothing beyond this can be said.

* * * * * *

We have now to deal with the differentiating points, and especially to note them as regards the intracranial portion of the system.

I propose to discuss the matter, so far as necessary, under the following headings:—

1. Traumatism.
2. Functional troubles.
3. Ordinary organic lesions.

1. Traumatism

Speaking generally, the incidence of an accident of gross nature and its immediate results are easy enough to appreciate, but there are a few cases of nerve troubles not perhaps usually included in the term, but which, I think, should be so included, for they are essentially traumatic in constitution. Chorea after fright, neuritis or so-called occupation neurosis, *i.e.* after prolonged special use of nerves, and the results of shock, are illustrations of my meaning.

Probably in all there may be an underlying functional or structural weakness of the nervous system, but this does not really affect my point.

If we thus widen our conception of traumatism, we have the following separate groups of possible results:—

(*a*) Gross laceration or bruising of tissue, with acute inflammation, followed or not by suppuration and abscess formation, of all of which the traumatism, with entry of pyogenic germs, is at once an obvious, complete, and satisfactory diagnostic explanation.

(*b*) Tumour of malignant or simple character, where again nothing more need be said, for the tumour is either a blood clot, or it remains in the shape of a cyst, or is of a heteroplastic nature — there are too many cases on record of the last incident to leave any room for discussion as to the sequence of events. (For further notes, *vide* p. 323.)

(*c*) Sickness or degeneration of neuron processes, which may end in recovery, or in ultimate death and sclerosis of the whole neuron, according to the severity of the changes started in it and the general recuperative power of the animal functions of the whole body.

This is, I think, the explanation of such cases as those mentioned above—shock, chorea, occupation neurosis—and possibly others. I believe that the potential (to use an electric analogy) of the stimuli sent through the neuron processes has been so high, or the current so long continued, as to have heated, melted, or disturbed the molecular constitution of the wires or connections, with resultant temporary or permanent loss of conducting power. This analogy must not be driven too close in detail, but in broad outline seems to me to offer the best explanation of such cases. However this may be, the relationship of external cause with the clinical symptoms is admitted by all. Of the diagnosis of chorea and shock little needs to be said; their features are unmistakeable. The only problem connected with them is whether the connections or wires of the nerve mechanisms can be repaired, and this point can only be proved by time. Suffice it to say, that repair is the rule, though this may be not quite complete. Of the identification of an occupation neurosis or neuritis one can only emphasise the great fact, from which, indeed, they derive their name, viz. that the pain or spasm, or inco-ordination, complained of by the patient is only pro-

duced by the special movements peculiar to the particular occupation (writing, violin playing, etc.). None of them appear when the same nerves and muscles are used for other combinations. This is at least true in the early stages of the malady, but recent observations tend to show that a definite neuritis does frequently exist. If and when this is so, it is easily explained on the above hypothesis. The fact that these neuroses are exhibited only in the performance of one special combination of movements is a strong argument in favour of a primary lesion in the cortex, where combined movements and not muscles are chiefly represented (*vide supra*, p. 257). It may also be mentioned that the following, amongst others, suggest a cortical seat for the troubles causing chorea and shock :—

(*a*) That they are practically always associated with some alteration in the mental or psychological conditions of the patient.

(*b*) That they very frequently exhibit themselves on one side of the body only.

(*c*) That the trouble is exhibited by complex combined movements which are originated in the cortex.

2. Functional Troubles

On p. 275 will be found a definition of the term "functional," and the tables and remarks throughout this section may be used to locate the weakened neurons, for it is only by perverted function as exhibited peripherally that we can locate the weakness. Here I wish to draw attention to a division of these cases into (*a*) those due to traumatism as defined above, and (*b*) those due to a general constitutional cause resulting in a loss or perversion of will power. In the latter group of cases, constituting the hysteria of the layman, or the "cussedness of the individual," I believe that the primary and essential lesion resides in the cortex of the brain (? and cerebellum), and consists in imperfections of the synapses of the internuncial neurons with one another, perhaps associated with altered stability of the molecular constitution of the neurons themselves.

3. Ordinary Organic Lesions

There is one exceedingly important general observation to be made which links all these into one. It is this: We must never forget the great frequency with which acute vascular lesions are found as a complication—very frequently forming the closing episode—in

the more chronic troubles, particularly in tumours and in sclerosing lesions; so that, while recognising the acute condition, we must never omit to inquire very carefully for evidence of a possible pre-existing chronic trouble (*vide* also pp. 272 and 278). By putting the definite question, "When was the patient last quite well and in his normal condition?" and further, by inquiring for any slighter manifestations of ill-health that may have preceded the actual attack for which our assistance is required, we shall not only get evidence of a second lesion if one exists, but we shall also gain information that may be of the greatest service in deciding between the various possible causes of the attack.

We may now proceed to an analysis of the lesions themselves individually. Those which I propose to discuss briefly are:—

Inflammation, hæmorrhage, thrombosis, and embolism (acute vascular lesions).
Idiopathic affections of neurons.
Tumours.

The following analysis, with the appropriate change in locality, will frequently apply as well to the cord and nerves as to the brain.

Broadly speaking, they divide themselves clinically into two groups: (1) irritative, (2) destructive lesions; but the distinction, though useful, cannot be maintained in its entirety. For in the life history of an irritative *pathological* lesion (physiological stimuli apparently have no such limits) there is to be noted, first, a period of excessive functioning in the affected neurons (pain, paræsthesiæ, twitchings or contractions of muscles), followed by a period in which this excess becomes less and less, until it becomes a negative quantity, finally function is completely abolished (anæsthesia and paralysis), in which stage the symptoms are those of a paralysing lesion—another reason for carefully ascertaining the whole history of the case. Then, again, the distinction of irritation *v.* destruction will depend in some cases on the situation of the lesion, meninges, or perineurium *v.* bulk of brain, cord or nerve strands. Inflammation, hæmorrhage, and frequently also tumour, will at all times offer good examples of this variability with locality.

Acute Vascular Lesions

Acute Inflammation.

(For diagnosis of Peripheral Neuritis, *vide* pp. 281 *et seq.* and pp. 292 *et seq.*)

As it attacks the central nervous system it occurs in two well-

marked forms, *i.e.* meningitis and encephalitis or myelitis, which require separate notice.

Of the Membranes, Meningitis.—Here, as anywhere else, acute inflammation excites general symptoms, viz. pyrexia, with malaise and discomfort. The special points leading us to locate it in the nervous system are the concentration of the pain in the back, or in the head, with rapid onset of grave symptoms of implication of nerve matter, or of pressure on it (affections of cranial nerves—squint, facial paralysis, etc.—vomiting, optic neuritis, unconsciousness, jumping of muscles, feebleness of gait, or paraplegia, etc.). In its irritative stage it may require separation from meningeal hæmorrhage or tumour. From the former, its slower onset and the pyrexia will be sufficient; from the latter, the pyrexia again and its more rapid (a day or two at most *v.* a week or two at least) onset should usually leave no doubt.

In its paralytic stages it becomes essentially a cortical myelitis or cerebritis, and only the history shows that such began as a meningitis.

Of the Cord.—The rapid onset of a complete paraplegia could only arouse suspicion of blocking or rupture of a vessel apart from a myelitis. Thrombosis or plugging of spinal vessels is so rare as to be a mere curiosity, but both this and hæmorrhage will be in their earlier stages sufficiently separated from myelitis by their absolutely sudden onset, and by the absence of pyrexia. In their later stages all three lesions would be identical.

Of the Brain.—A primary cortical acute cerebritis is extraordinarily rare. If it occurred it would precisely resemble an acute meningitis, except that there would be a more rapid onset of paralytic phenomena, especially in the limbs (vertical meningitis), or in cranial nerves (basal meningitis).

Acute primary medullary cerebritis, *i.e.* in the mass of the brain, is practically only known in the shape of an abscess, and then the principal point is to separate it from a tumour. Their likenesses and differences may be summed up thus. Both will be equally likely to have—

1. Vomiting.
2. Optic neuritis.
3. Headache (intense).
4. Localising paralytic phenomena, or irritative.

On the other hand, they will probably differ as follows:—

Abscess.	Tumour of other Nature.
1. A history of some peripheral suppuration—ear, nose, and other discharges—which has possibly ceased just previous to symptoms of cerebral trouble.	Probably no such history, but there may be a tumour elsewhere suggestive of a primary growth, to which the cerebral one may be secondary.
2. Symptoms may quite possibly come on with great rapidity, and rapidly get more serious.	Symptoms rarely increase with great rapidity unless it be with instantaneous exacerbation, indicating hæmorrhage into tumour.
3. Temperature a little raised, or more probably subnormal.	Temperature either quite normal, or with greater and more irregular ranges than in abscess.
4. Irritative phenomena rare, paralytic phenomena at once or rapidly coming on.	Irritative phenomena commoner, and lasting longer than in abscess.

Hæmorrhage, Thrombosis, and Embolism—

The one essential feature common to all these, and the one by which they are as a group separated from all other lesions, is the instantaneous rapidity with which their symptoms commence, and speedily reach an acme. The two latter never as a simple diagnosable lesion occur in the meninges, but the former frequently does so, and it is of some importance to distinguish this situation of it if we can:—

Meningeal Hæmorrhage.	Hæmorrhage into Bulk of Brain or Cord.
1. Commoner when idiopathic in young subjects.	Commoner in older subjects when without exciting cause.
2. Very rare, except as the result of traumatism, forceps at birth, *e.g.*, or blows on the head or back.	Very common, without any history of traumatism: is equally rare as the result of violence, unless this be very severe.
3. Commencement of symptoms sudden, but generally some minutes or even hours before they reach their acme, *i.e.* more ingravescent.	Instantaneous commencement, and acme reached almost with the same rapidity; gradual ingravescence is much rarer, though well recognised as occasionally occurring.

In this point of contrast it is obvious that exceptions must occur in connection with the size of the vessel which has ruptured, *e.g.* the middle meningeal or the basilar, on the one hand, the symptoms of which will be indistinguishable almost from intracerebral hæmorrhage; or, on the other hand, a very small branch of an intracerebral artery, the symptoms of which may be very slowly ingravescent. Concussion, or brain bruising, is another exception, but its onset is always traumatic and sudden.

	Meningeal Hæmorrhage.	Hæmorrhage into Bulk of Brain or Cord.
4.	The symptoms usually go through an irritative stage, which is easily observable and may be prolonged (pain or convulsive movements).	The symptoms cannot be said to have any irritative stage; they are destructive from the earliest time of observation.
5.	The subsequent course may show complete recovery, though possibly an irritative focus may be left behind.	Recovery never complete, and the *focal* lesion will always be found to be a destructive one.

In discussing the separation of the three troubles as they occur in the bulk of the brain or cord, it must be admitted once for all that a positively certain diagnosis (ante-mortem) is impossible, but a more or less intelligent and probably correct guess may be hazarded by attention to the following points:—

1. That in the spinal cord the communication of the vessels is so free and easy that embolism and thrombosis are of the very rarest occurrence of sufficient extent to cause a definite paraplegia.

2. *Age.*—Though the cause leading to embolus direct from the heart (acute endocarditis) is commoner in young subjects, and also syphilis and tubercle as active causes of local thrombosis, yet none of them cease absolutely as age advances. Moreover, atheroma, another principal cause of thrombosis or embolism (from debris), is distinctly an affection of advancing years. Hence age, *per se*, gives us no very strong and leading guidance; nevertheless it must be stated that, *ceteris paribus* (which they seldom are), the older the patient the more do our thoughts go in the direction of hæmorrhage.

3. Marked premonitory symptoms of slight attacks are likely to make us think of thrombosis, especially if syphilis is present, or tubercle a probability. The reason for this is that preliminary small hæmorrhages are rare, and so also, I believe, are small

emboli, while as an artery gets gradually obliterated we should expect premonitory evidences of disturbed nutrition in areas which are ultimately going to die.

4. *State of Consciousness.*—If we bear in mind that hæmorrhage forces asunder (by a pressure increasing as the cube of the radius of its bulk) the fibres of the brain, and adds materially to the bulk of the intracranial contents by blood which should be escaping by the veins, thus increasing the intracranial pressure; if, too, we bear in mind that loss of consciousness (under the circumstances we are now considering) is probably due to increase of pressure on the cerebral cortex, or to violent impressions upon it, and then consider that the plugging of a vessel from any cause has no such tendency, we shall see the probable explanation of Trousseau's statement (which is, I believe, still in accord with clinical experience), that "Whenever hemiplegia, complete and absolute" (therefore due to a rather serious lesion), "occurs suddenly" (therefore due to vascular lesion) "without the loss of consciousness" (therefore without much pressure), "it is more likely to be due to softening (plugging) than to hæmorrhage."

5. *State of Heart.*—If in a young subject, otherwise in usual health, we hear cardiac bruits, they will be, *ipso facto*, suggestive of embolus, provided the heart is acting fairly quietly and regularly, and the more suggestive the more recently the bruits are known to have developed. If, on the other hand, we find a powerful heart acting violently with or without bruits, we suspect that this power has ruptured a weakened vessel, especially if the attack (hemiplegia) came on during exertion or excitement (one of the great dangers of alcohol when taken by a person with damaged vessels). Finally, if the heart beat is very feeble, as after a severe illness of any sort, and during convalescence, the feebleness of the circulation is suggestive of thrombosis, either locally at a distance from the heart, *i.e.* in the brain, or of clotting in the irregular chambers of the heart and propulsion of an embolus thence.

6. *Temperature.*—Hæmorrhage always causes a fall in the temperature, which may continue till death or be replaced by irregular pyrexia. Plugging of a vessel rarely causes much fall, and, if it does so, the temperature soon rises to normal, and continues there with very slight or no variation, so that the greater the excursions of the thermometer the more do we think of hæmorrhage.

If these various indications are properly balanced, a correct conclusion will more often than not be arrived at, though many mistaken diagnoses will undoubtedly occur.

It must not be overlooked on the post-mortem table that a fatal plugging may yield no very obvious signs, because the softening which we look for as proof of plugging is a phenomenon which only commences some little time after the circulation through the part has ceased. It may not be out of place, too, to mention that, whether the softening be white, red, or yellow, it is merely an accidental result of (1) the age of the process; (2) the amount of blood or other pigment, or their derivatives, left in the area by circumstances of absorption; it has no special significance suggestive of a difference in the pathology of the lesion.

Idiopathic Affections of Neurons

Though in no sense of the word idiopathic, there is no other connection in which the *effects* of hæmorrhage or plugging can be more appropriately placed; consequently, we must tabulate the above heading as follows:—

1. Secondary to their death by gross violence, whether from hæmorrhage or plugging.
2. Idiopathic—
   - (*a*) Acute, usually termed acute inflammation.
   - (*b*) Chronic, usually termed primary sclerosis.

1. Of the secondary form but little need be said; it undoubtedly occurs as a sclerosis of a tract, or track, after hæmorrhage from or plugging of a vessel has destroyed the nutritive mechanism, but it adds little or nothing to the clinical picture already portrayed. The paralysis, or anæsthesia, or both, and the exaggerated reflexes appear rapidly as the result of the death of the neuron; the subsequent sclerosis merely fixes these in rigid features, and adds certain confirmation to a previous diagnosis of organic mischief.

2 (*a*). The acute primary affection has its chief, if not its only, seat in the neuron cells of the motor areas (nuclei of nerves), at any rate this localisation property is so constant that clinically it affords one of the strongest points of evidence in favour of the particular pathological lesion, *i.e.* of primary acute inflammation or degeneration. We find it most commonly in the spinal cord as acute anterior poliomyelitis, the diagnostic points of which have already been considered on p. 292, but it must not be forgotten that the nuclei of origin of the motor cranial nerves are absolutely analogous in function, if not even serially homologous in structure, to those of the anterior cornua, and are therefore found to be

liable to the same affections; hence an acute bulbar paralysis or an acute affection of an isolated cranial nerve nucleus may and does occur. Its history will be one of acute (*i.e.* complete within twenty-four hours) onset, accompanied probably by a slight pyrexia; the latter may be so slight or fugitive as to escape observation, and hence the disease may resemble at its onset a small hæmorrhage or softening. The differences in such an acute case will principally consist in the following:—

(*a*) Although it is possible, yet as a matter of clinical experience, the onset rarely is so instantaneous as in hæmorrhage.
(*b*) There will be at any rate in a few hours after onset an almost complete absence of any sensory phenomena.
(*c*) In the bulb if the cause of the trouble were a hæmorrhage or softening, it is almost inevitable but that some limb paralysis or anæsthesia should be found, for the vessels supplying these motor nuclei do not confine their nutritive supply to these alone; and we have already noted the peculiar precision with which this lesion does pick out the motor cells only.

In the identity of its results upon the peripheral nerve and the muscles supplied by it (degeneration and atrophy), it has to be separated from purely peripheral lesions; difficulties arise here more in theory than in practice; nuclear lesions are seldom quite complete (*e.g.* of third, seventh, sixth, twelfth, etc.), while peripheral ones are specially prone to be so, particularly if the nerve (*e.g.* the seventh) passes through a bony canal. Again, if the peripheral lesion be only partial, its causes (meningitis, gummata, tumours, etc.) are associated with general symptoms or local phenomena (swelling, pain, etc.) of such obtrusiveness as to leave little room for doubt if the case be seen in an early stage. In later stages diagnosis will be impossible without the history to guide us.

2 (*b*). In the chronic form of primary sclerosis of tracts, of which the following are the chief types:—

Tabes dorsalis
Lateral sclerosis
Progressive muscular atrophy
} of the cord;
Disseminated sclerosis of cord and brain;
Chronic bulbar paralysis of bulb;
? Paralysis agitans
? Senile tremors
} of brain cortex;

the principal feature which separates them at once from every other lesion — except a very slowly growing tumour — is the extreme chronicity of the process, so that symptoms are slowly developing for many months at least, and often even for years. The extreme precision with which this affection, *i.e.* primary sclerosis, picks out certain tracks and strands (except disseminated), is also as marked a feature as in the acute form, so that, when combined with chronicity, it leaves little room for doubt, at any rate in advanced cases. The individual diseases have already received (*supra*) sufficient attention; it remains here only to remark on the likeness of the process to functional disorders. If we consider the minute pathological processes underlying the gross sclerosis (the final stage), it is easy to see that in the early stages of the trouble we are now considering, it is probable that a time will occur in which only function will have suffered, and consequently this will be a stage in which the greatest difficulties may occur in separation; in fact, there may be, and even probably must be, identity of symptoms with a difference only in future progress. Attention may be drawn to p. 297, where the differences between disseminated sclerosis and functional troubles are discussed. Besides what is there recorded there is only the general consideration of the element of time; this, in the beginning of a simple functional trouble, which is not going to proceed to sclerosis, is likely to show confusion in many systems, as opposed to stress on one system in a case that is going to end in sclerosis; and then, again, later will show a stationary or improving condition in the former versus a downhill course if the fundamental meaning of the symptoms is the starting of a progressive process of decay and degeneration.

Tumours of Brain (and of Cord)

By their comparative chronicity or comparative acuteness, according to the point of view from which we regard them, these troubles separate themselves almost conclusively from vascular and acute inflammatory lesions on the one hand, and, on the other, from chronic idiopathic scleroses. From the latter, too, they are also separated by the fact that they are very indiscriminate in the pressure they exert: they spare nothing that comes within their sphere of influence (hence the predominance in the cord of root symptoms). As they affect the cord they will receive no further notice, but from their importance in the recent advances of cerebral pathology and treatment they must be discussed a little more fully as they affect intracranial structures.

It is common knowledge that the tripod on which their diagnosis mainly rests has for its three legs:—

Headache.
Vomiting.
Optic neuritis.

To these are frequently enough added some specific features indicating its locality, and occasionally its precise nature. For the former, *vide* Jacksonian Epilepsy and the Cranial Nerves; of the latter it is only necessary to mention a definite history of syphilis, tubercle in the choroid or elsewhere, and a primary focus in some organ of sarcoma or carcinoma.

Now, any one of these three legs may be permanently knocked away and yet the diagnosis stand, while even two may be removed for some time without a complete overthrow of the diagnostic superstructure; but if all three be gone it is only in the rarest of instances, and more by inspiration than logic, that we should venture to assert that a cerebral tumour was present because of some incidental localising suggestions.

Headache.—Pain in the head, even severe, is such a common complaint that not much stress can be laid on this symptom until the other common sources of it have been excluded. We may say that unless at least one of the other two guiding indications were present it would hardly arouse suspicion of a cerebral neoplasm (not but that the neoplasm may be there, without our suspecting it), except it possessed in rather a marked degree some or all of the following features: (1) very persistent and resistant to remedies, and possibly paroxysmal; (2) so severe as to keep the patient awake at night; this should be made a very strong point, as patients vary very much in their estimation of the intensity of a headache; (3) located in one, and that perhaps an unusual part of the head; (4) often associated with acute additional pain or tenderness on percussing the head in that region (this is commoner in superficial tumours, and when definitely present is a valuable localising sign).

Vomiting.—If of cerebral origin (*vide* pp. 158 and 159) is usually stated to be unassociated with much, or any, nausea; totally independent of food (often attempted with an empty stomach), and of a peculiarly forcible (pumping) character; certainly none of these features is absolutely constant, even in the certain presence of a tumour, but we may safely say that when a patient makes special complaint of vomiting as the chief symptom, and no definite gastric or alimentary trouble can be found to account for it, it is imperative

upon us to examine the fundi oculorum and inquire more particularly about the headache. The diseases likely to cause such vomiting of remote or obscure origin are tabes dorsalis and Addison's disease, neither of which should offer any difficulties in exclusion, at least from the possible occurrence of a tumour.

Optic Neuritis.—Inasmuch as a definite optic neuritis occurring as a totally independent disease, apart from any discoverable causation, is the very rarest of phenomena, if not absolutely unknown, it necessarily follows that if we can establish its certain presence, and if we can exclude other causes, we have almost proved to a demonstration the presence of gross intracranial mischief of but few categories.

As regards its recognition it cannot be too strongly insisted upon, nor too often repeated, that severe optic neuritis and other pathological changes in the fundus may be present without the slightest voluntary complaint on the part of the patient of subjective disturbances of vision, nay more, he may deny such even when the direct question is put to him, so that the ophthalmoscope is absolutely necessary for diagnosis.

The following is a tolerably complete list of causes of the changes found in the fundus, with the main characters of separation :—

Gout, cirrhotic kidneys, albuminuria.	Hæmorrhages, or white atrophic spots, the chief feature in the fundus ; the examination of the urine, and other evidences of vascular degeneration, leave no room for mistakes.
Specific fevers, typhoid, *e.g.*	The pyrexia, the rash, and general symptoms too well marked ; the neuritis itself may be indistinguishable.
Leucocythæmia and purpura.	Again hæmorrhages are the prominent feature in the fundus ; examination of blood and enlarged spleen in the one case, and hæmorrhages in the skin in the other, are sufficient to prevent mistakes.
Embolism of central artery.	Changes are in the yellow spot, not in the disc, except as atrophy ; arteries and veins small instead of large. Loss of sight probably complete in the eye affected.
Anæmia.	Optic neuritis rare, except in cases of very marked pallor ; examination of blood gives certain information. I have known even Dr. Jackson deceived in such a case, however, and diagnose tumour from the headache and optic neuritis when they were dependent upon simple anæmia.

Lead poisoning.	It is very rare that optic neuritis is the only sign of plumbism in a doubtful case; wrist drop or colic, or blue line, nearly sure to be present; occupation or opportunities for absorption important.
Errors of refraction.	This is rather a pitfall in estimating the presence of a neuritis than a definite cause of that condition. They must be carefully excluded as the possible causes of visible fundal peculiarities before other ones are sought; and the results of their correction noted.
Tubercle.	Visible as small nodules in the choroid; their presence is absolutely conclusive as to the nature of a tumour or a meningitis if previously doubtful, but they are only rarely present even when tubercle is the cause we are seeking for certain symptoms.
In tumour of ordinary character, or syphilitic or tubercular, and in abscess or meningitis of any causation.	The optic neuritis when present has usually no distinctive characters peculiar to each (? tubercle above), but is very frequently present with any of them as very strong evidence of a gross diagnosis.

Such are the principal means we have for a pathological diagnosis in general terms. We have already considered the separation of meningitis and also of abscess. The question of the space of time during which a lesion is developing its features has already been repeatedly alluded to, and so also have those features which assist us in forming an opinion as to the precise locality of a disturbing factor. The question cannot be pursued farther without going into very special discussion unsuitable for the present work.

CHAPTER IX

URGENCY CASES

UNDER this heading I propose to discuss the diagnosis of a very mixed group of cases with which the daily press has from time to time made and kept us familiar under the attractive (?) title of "Drunk or Dying," in allusion to the fact that so many of them, while ending fatally, have a very close resemblance to the varied phases of alcoholism in its numerous stages.

It is for lack of some definite principles in diagnosis (and treatment) that so many mistakes have occurred, mistakes from which no extrication is possible, and through which more than one medical reputation has foundered, and public institutions have had much undeserved censure cast upon them.

We may define the group to which I refer as "Cases in which we are called upon—usually in a great hurry—to examine and treat a patient in whom *sudden* and apparently severe illness has supervened, and whom we have not been previously attending, or with whose previous history we are at the time unacquainted." This last condition is put in to temporarily exclude cases of illness which are known to be complicated with sudden storms, though the following analysis will show that such are not usually present.

We will commence with a bare enumeration of the lesions which are liable to take place suddenly, or, perhaps more appropriately to our subject, are liable to cause sudden symptoms in persons who are apparently in perfect health, or at least are able to go about the public thoroughfares unattended. They divide themselves fairly naturally into groups as follows:—

I. Hæmorrhage.
II. Vascular lesions other than hæmorrhage.

III. Traumatism.
IV. Sudden death.
V. Lesions and conditions of the nervous system.
VI. Poisoning.

This is a fairly complete list of "urgency cases." It may seem a very formidable one to carry in mind, but in practice it is not so, for many cases are self obvious from the first, and early treatment can proceed on but very few lines.

Group I.—HÆMORRHAGE

(*a*) Through breach of skin.	*e.g.* Traumatism, varicose veins, etc.
(*b*) Appearing externally through rupture of vessel internally.	*e.g.* Hæmatemesis, hæmoptysis, epistaxis, or hæmorrhage from rectum.
(*c*) Concealed hæmorrhage.	*e.g.* Rupture of aneurysm, or of tubal gestation, or of a vascular organ, especially spleen; hæmorrhage into stomach without vomiting, or into intestine without appearing in a stool. (Cerebral hæmorrhage is not included in this group, as it is brain laceration and compression that cause its symptoms.)

From the point of view of a provisional or preliminary diagnosis guiding us to first or immediate treatment, this group stands almost by itself, and may at once be dismissed rather summarily. Subdivisions (*a*) and (*b*) are at once obvious by the blood round the patient (if the body has been removed or the blood cleaned up there will obviously be some history of such having been done). Group (*c*) offers a little more difficulty, but here it is fair to assume that if the hæmorrhage is sufficiently severe to necessitate the attendance of a medical man, it will be sufficient to cause very marked blanching of the face, and faintness, with the small pulse, weak or husky voice, and sighing respiration characteristic of a severe loss of blood. A ruptured aneurysm, spleen, or ectopic gestation is, besides the faintness and blanching, also very likely to cause severe local pain, directing attention to the region in question.

Group II.—Vascular Lesions other than Hæmorrhage

e.g. Simple fainting, angina pectoris. (Blocking of blood-vessels is excluded here, as the symptoms entirely depend on the locality of the block.)

A diagnosis of either of these conditions is usually, like that of hæmorrhage, fairly easy. Either of them may have ended in rapid death (a subject of special grouping, *vide* below), in which case let me strongly urge that no diagnosis should be given without a complete autopsy. Fainting, if not due to actual hæmorrhage (*vide* above), is commonly very transient; if prolonged, it can only be diagnosed as a simple lesion by exclusion of the indications of Groups V. and VI. (*vide* pp. 331, 332).

Angina, if of any severity (and it is only then that it becomes an "urgency case"), will be recognised by the position of the pain in the chest, and the fixed attitude of the patient from the fear of death on movement.

Group III.—Traumatism

The only conditions that can arise from traumatism are—

(1) The actual injury to limbs or viscera.
(2) Shock to the nervous system, which may be sufficiently severe to cause death.
(3) Unconsciousness, from—
    (*a*) Shock, pure and simple,
    (*b*) The nature or locality of the injury, *e.g.* of brain.
(4) Death.

If the victim is conscious, and thus a history obtainable, no difficulties in forming a preliminary diagnosis can arise, and we are not here concerned with anything deeper or more accurate, the discussion of which indeed involves a whole treatise on traumatic surgery. If, on the other hand, the victim is unconscious though not dead (again *vide* Sudden Death), and no history obtainable from friends or bystanders, the patient must be carefully examined for any signs of injury (fractured base of skull, or severe crush, bullet wounds, etc.), and then, should nothing be found to account for the unconsciousness, the case must temporarily be placed as "unconsciousness" amongst the group of nervous lesions, and its diagnosis sought by means of the indications given under

that heading. The position of the body, as at the bottom of a ladder or stairs, etc., may suggest a fall, but even if the fall be proved, it may still be due to epilepsy, apoplexy, etc. The shock of traumatism, especially if the latter be not very severe, may take the shape of a mental condition closely resembling alcoholism (*q.v.*).

Group IV.—Sudden Death

The interest of this group is purely medico-legal. There can be no clinical diagnostic problem involved, unless it be to give a warning to friends as to the possible sudden death of any patient, so that worldly affairs may be put in order in time. It is, however, impossible to ignore the condition in an article on "Emergency Cases," and so I have inserted the following table, which is but slightly altered from Professor Dixon Mann's article in the *Lancet* for June 26, 1897; the great assistance of which I wish to acknowledge. Dr. Mann states that in a case of sudden death one of three conditions will be found on autopsy:—

1. A recognisable disease or condition known to be commonly associated with a sudden end.
2. A recognisable disease, but which is not usually terminated suddenly.
3. No recognisable pathological condition anywhere in the body.

Tabulated and arranged, with a few additions and alterations, this first list gives the following:—

(*a*) Morbus cordis (in about half the cases.—Mann).	Valvular disease.
	Possibly congenital abnormality.
	Atheroma of coronary arteries (with or without history of angina pectoris).
	Fatty heart.
	Sclerosis of muscle.
	Rupture of wall of a cavity.
(*b*) Hæmorrhage.	Ruptured aneurysm.
	Ruptured spleen.
	Into pancreas.
	Into brain (apoplexy).
	Into stomach or intestine.
	Ectopic gestation.
	Varicose veins, internal or external.
	Traumatism of a large vessel.

(*c*) Asphyxia, with a condition or disease known to produce it rapidly.	Air, serum, blood, or pus in pleural cavity. Œdema of, or membrane in, glottis or polypoid growth. Pressure of tumour (solid or liquid) on larynx or trachea. Embolus of air in a vein, or clot in a pulmonary artery. Blood, or other vomited or swallowed foreign body in air passages.
(*d*) Miscellaneous group, in which precise mode of death not obvious, though the morbid condition is easily recognisable, and is known on occasions to cause very rapid death.	Perforation or strangulation of gut, and peritonitis. Exophthalmic goitre. Addison's disease. Nephritis of any kind. Pneumonia, especially in old people. Abscess or tumour of brain. Meningitis. Obvious damage to a viscus from traumatism.

In his second list Professor Mann includes some of the conditions I have put in the first one, and also the following:—

Diphtheria, without membrane being the apparent cause; probably neuritis.
Phthisis, with exhaustion probably.
Ulcerative endocarditis; may be signs of septic infarction.
Pericarditis, with effusion of serum or pus.
Adherent pericardium, probably with recognisable changes in the muscle of the heart.
Gall-stone colic } May find the stone in the duct or ureter,
Nephritic colic } but it may have slipped back or out.
Gout, especially in corpulent individuals, recognised probably by the cirrhotic kidney corroborating the joints or tophi.
Hydatid tumour, which has ruptured.

The third list of Professor Mann's I have enlarged by including in it several conditions which he has placed in the first and second; my reason for so doing is that it is only in very exceptional circumstances that the results of the autopsy are sufficient for a diagnosis; there is required in addition a reliable history of the patient in some form, or a witnessing of the last scene. It includes:—

Asthma—possible signs of asphyxia.
Pertussis—possible signs of asphyxia.
Overlaying of infants.
Epilepsy—possible signs of asphyxia;
Convulsions in children;
Spasm of glottis—possible signs of asphyxia;
(Laryngismus stridulus);
} and possibly clenching of hands.
Acute rheumatism—cardiac syncope.
Zymotic diseases—occasionally by intensity of the poisoning—without a rash.
Hyperpyrexia—sunstroke, acute rheumatism, etc.
Influenza.
Excitement or fright as in quarrelling, or near approach of any danger—thought of an operation, etc.
Chronic alcoholism—probably some neuritis of pneumogastric.

On these lists I do not propose to say anything further; their importance is obvious and enormous; but when we have admitted the fact of sudden death, and have made a thoroughly careful post-mortem, clinical medicine ceases its functions, and legal procedures come to the front with all the available evidence as to what was happening, or had just happened, when death took place.

We now come to the two groups which include the real crux of the position, and are the essential clinical elements in drunk or dying. They must be considered in some considerable detail.

Group V.—Lesions and Conditions of the Nervous System

Cerebral plugging or rupture of a vessel, constituting apoplexy of the laity.
Epilepsy—including post-epileptic states, and also malingering of fits.
Hysteria.
Plumbism—lead encephalopathy.
Lunacy—especially G.P.I.
Cerebral tumour—with epileptiform convulsions.
Sunstroke.
Meningitis, and acute encephalitis.
(Simple fainting will be here considered; as its main symptom is apparently cerebral.)

Group VI.—Poisoning, including two Sub-Groups

A. Autogenetic. Uræmia.
Diabetic coma.

B. From without, which may include any and every poison, however administered, but from our present point of view has especial reference to—

Alcohol.
Opium and other narcotics, such as chloral.
Belladonna as a deliriant.
Strychnia as a convulsant.

Both for diagnosis and treatment it will be well to maintain a chronological order of events, as this will render the discussion much more practical, even if it introduces a little theoretical inconsistency.

Commencing then with the history. While getting ready to go or going with the messenger we must inquire into the particulars of the seizure as closely as possible: asking, for instance, Has the patient taken anything?—if so, Of what known nature?—Whence obtained?—Is he in a fit? etc. From the answers to these questions, if they be intelligent or even intelligible, valuable hints may be obtained. I am bound to admit that in the majority of cases there will be either no history at all, owing to the absence of witnesses, or the history will be unintelligible, owing to the anxiety, fright, or imbecility of the messenger; or, again, it may be purposely misleading in some few criminal cases. Such as it is, however, listen to it, and use your own judgment as to its value.

On arrival, the first thing is to clear the room or a space round the individual, to give him air, to prevent bystanders doing mischief, and to enable you to exert your own efforts without unnecessary interruption. Secondly, while doing this, you will have a few moments in which to note the general aspect of the patient. Is he motionless and apparently dead, or is he in a fit, convulsed and obviously living? Is his face natural in colour, or is it blue with livid lips, and is froth issuing from them?—if so, Is the froth blood-stained? Is he sweating profusely, or is his face dry? Thirdly, while noticing these facts loosen everything from the neck, cutting the clothes if necessary for rapid action to prevent imminent asphyxia. And lastly, if convulsed, restrain him in some way, so as to prevent him from injuring himself by the violence of his movements.

We are now in a position to take diagnosis into consideration, and this obviously proceeds in two directions, according to whether the patient has convulsions and is living, or whether he is quiet and possibly dead.

If he is in a "fit," this may be due to—

Cerebral (hæmorrhage, tumour, etc.);
Epilepsy;
Hysteria;
Malingering;
Poisoning—strychnine especially, but possibly opium or other poison;
Uræmia;
Dentition, etc., in babies;
Tetanus;

and the following are the points most worth attention to decide which of these is present.

1. *Age and Sex.*—Of age little need be said, for at all ages many things are possible. It is obvious that dentition can only occur in babies, and in these young subjects convulsions are very common from every form of reflex irritation; the precise cause can only be cleared up by the same careful examination as in adults; the condition, though very common, hardly gives rise to difficulties in our present connection, *i.e.* with "urgency." Sex, too, has only this influence, that in females, and especially from seventeen to forty years of age, we lean more strongly to a diagnosis of functional trouble of no great immediate danger.

2. *The Eyes.*—A most, perhaps *the* most, important seat of clues.

Are they open or shut?—if shut, Is there much resistance to your opening them by lifting the lids?—Is the patient looking round furtively? This furtive glancing and resistance to opening the eyes is very suggestive indeed of hysteria or malingering. Then the state of the pupils, note are they equal or unequal, dilated or contracted, do they react to light or not, is the conjunctival reflex present. In epilepsy the pupils are dilated and insensible to light, or occasionally oscillating without obvious cause: in hysteria they react readily to light, and in malingering (unless artificially tampered with) they will obviously do the same. In poisoning they will usually not act to light, if it be by strychnine, the only likely convulsant, they will be dilated, as will also happen if asphyxial convulsions are due to some poison; should it happen to be a case of opium poisoning with

convulsions they will be contracted. In uræmia the pupils are as a rule fixed and dilated, they react very slowly, if at all, to light, but it may happen that they are of natural size. If the pupils are unequal we shall think of pontine or other cerebral hæmorrhage, or of general paralysis of the insane. The conjunctival reflex will be absent in epilepsy, uræmia, and other more dangerous conditions; certainly present in hysteria and malingering.

We may sum up the matter by saying that if (1) fixed (small or large), or (2) irregular and unequal, pupils are present, or if (3) the conjunctival reflex is absent, then the condition is serious, and may be a dangerous one. If, on the other hand, the pupils react readily to light, it is probably a less serious matter, and if the conjunctival reflex is present this adds greatly to the probability of a recoverable condition.

3. *Froth coming from Mouth.* — This is usually present in epilepsy, and very possibly blood-stained from biting of the tongue. If it be blood-stained, epilepsy is certainly most probable in the absence of motive for malingering. Malingerers will use soap for froth-making purposes, so that the mouth must be examined for this article. Froth may be present in poisoning, but has then no special significance except as suggesting hydrocyanic acid, but then fits are not present.

4. *Note the Nature of the Convulsions.*—They are most exaggerated perhaps in hysteria, and most universal. The malingerer is apt to over- or under-do the movements. In poisoning by strychnine an attack is soon over and followed by complete relaxation of muscles; in tetanus this relaxation is incomplete; moreover strychnine convulsions are more marked in head and trunk (opisthotonos or emprosthotonos), while tetanus is more likely to be concentrated in the jaws and neck. In epilepsy, too, opisthotonos is usually absent, and malingerers cannot keep it up for long, even if they can assume the position at all. In cerebral hæmorrhage or blocking with convulsive movements, these will take the shape of conjugate deviation of head and eyes, a most important sign, hardly ever met with except with such gross lesion.

5. *The General Appearance of the Face.*—In malingerers certainly, and usually in hysteria, the face is hot and flushed, and the body as well bathed in hot perspiration (note the state of the weather or atmospheric pressure) if the movements are at all violent. In epilepsy the face is possibly blue and congested, or more probably pale; it may be moist, but it will be with a *cold* sweat, and the same coldness will be present in poisoning. In uræmia the face

may be flushed, but it is more usually natural or pale without sweat.

6. *The General Consciousness and Capability of being Roused to Speech.*—In malingering and hysteria he can certainly be roused; in epilepsy and uræmia he cannot be; in poisoning he is either totally unconscious (hardly in this group), or is perfectly conscious and rational *between the fits.*

7. *The Pulse.*—Over this the patient can have no control. In malingering and hysteria the pulse will be full, bounding, and rapid from exertion. In uræmia and poisoning it is likely to be very small and rapid; in epilepsy it is usually somewhat accelerated; in gross cerebral lesions it is likely to be very slow, from cerebral compression.

Such are the most important means of immediate or rough diagnosis, none of them individually trustworthy; each has to be considered in relation to the others. I do not propose to consider the more accurate diagnosis any further, as the main object of this chapter is to draw a line between those cases for which a bucket of water or a police cell is advisable, and those for which skilled medical attention in a place of rest and quietude (bed at home, or in a hospital) is the only safe line to pursue.

We will now pass on to the second group of cases, viz. those in which convulsions with (genuine or feigned) unconsciousness are not present. Here the cases may be divided into three classes, though I shall speak of them only in two:—

I.—Those who are actively conscious (this consciousness may be very perverted in its judgment), and perhaps noisy and abusive, especially when undergoing physical examination.

II.—Those who are unconscious, but can be roused by speaking, pinching, pricking, etc.

III.—Those who are absolutely comatose, and cannot be roused at all.

Firstly, then, those who are actively conscious and may be noisy. Practically there are four things to be thought of here:—

(1) Alcoholism—drunkenness;
(2) Head injuries and cerebral disease;
(3) Lunacy;
(4) Poisoning other than alcohol—rarely;

and it must never be forgotten that the first may be combined with any of the others; a complication the possibility of which forms the principal crux of the problem.

Here again, the first thing is the history from others or from the patient, and it is very probable that in this class of cases it will throw strong light on the diagnosis, especially when taken with the manner in which it is told.

Lunacy, under these particular circumstances, is almost sure to betray itself either by the absolute nonsense the patient talks, or by the direct contradiction of, or lack of confirmation from, the story of those with him. The manner of relating his tale differs materially in the lunatic and the "drunk." The lunatic may relate his tale perfectly quietly and with pseudo-rationality, the drunken man is nearly sure (in this stage) to be very excitable and incoherent: the speech of the former will be complete in its individual words (except in general paralysis of the insane, when the long words may be slurred), that of the latter will have the easily recognised character of drunken speech.

If actual lunacy can be excluded, but we have a strong suspicion that alcohol has been at work with or without an accident of some kind, the tale of others may be of most material value. In cases of head trouble, whether from traumatism (shock is here an important element) or from disease, a little alcohol goes a long way, so that if there be the slightest suspicion of serious trouble, abuse and noisiness must be largely discounted, and the patient taken under professional care. A case in point occurred within my experience some years ago. The patient was at first quiet, but began to be noisy, abusive, and very pugnacious when examined. He was thought to be drunk, and was on the point of being sent away when another house physician saw him, thought he looked very ill and peculiar, and advised his admission to the wards: he died before reaching them, and the autopsy showed that it was a case of cerebro-spinal meningitis. See, therefore, how noisy and abusive conversation fits in with other facts in the history.

If what you hear is not sufficient, see what you can ascertain with your eyes. The colour and appearance of the face may help. In alcoholism it will be flushed; in head mischief, whether hæmorrhage or inflammation, the face is possibly flushed, but it is much more likely to be pale, or blue and congested—bloated; in a lunatic the face will seem natural, or possibly flushed with exertion; in deliriant poisoning the face will be dry and flushed, or cold and clammy. The condition of the pupils is as important here as in the case of convulsions; in alcoholism they are sure to be dilated, and probably fairly active; indeed, if the patient is not comatose from alcohol they are sure to be active. In head conditions,

though the pupils may be active, they are more likely to be fixed, dilated, contracted, or unequal, or at least sluggish in movement. In lunatics (except general paralysis, when they are unequal or sluggish) they are active enough. In poisoning in this stage they would be widely dilated and inactive, with brightly glistening eyes.

If still in doubt the temperature should be taken. This in serious cases is pretty sure to be markedly raised or lowered. If a normal temperature is found in a man who is actively conscious, or can be roused very easily, and if at the same time his pupils are active, the probabilities are that there is nothing very serious the matter. If, on the other hand, the temperature is raised, inflammatory conditions must be suspected, either in the brain or possibly elsewhere (unsuspected pneumonia, *e.g.*), and if markedly lowered we know that there is a depression likely enough to be or become dangerous, and the case had better be watched.

These points, if carefully considered, will be, as a rule, sufficient to prevent serious mistakes, but keep all your faculties on the stretch for any little suggestive point; finally, if in doubt, act as if the case were a serious one.

We will now pass on to the other group of cases—those in which the patient is quiet and actually unconscious, though he may be capable of being roused momentarily.

There is here only one golden rule of treatment: NEVER ALLOW AN UNCONSCIOUS PATIENT TO BE LEFT ALONE. If he is seen at a hospital take him in. Far better to take in fifty drunken men in one night, and let them lie on mattresses under supervision in a warm room, than let one die unattended. The one action can at the worst only subject you to mild chaff and cause temporary inconvenience, the other may ruin you or your hospital for ever. If seen elsewhere than at a hospital have him removed at once to a hospital or to his home (or some one else's), and do not leave him till some reliable person is in charge. This is a golden rule, from which no possible concurrence of circumstances should ever allow you to depart.

Accurate diagnosis is therefore, from the point of view of immediate treatment, but of little use, but for further treatment, and for our reputations' sake, we must endeavour to make one.

Any one of the conditions hitherto enumerated in this chapter may be present, or a combination of two or more. A drunken man, for instance, is not absolutely free from the chance of being injured by blows or falls, and he is even more likely than a sober one, *ceteris paribus*, to have an intracranial hæmorrhage, for if a man's

vessels are diseased and he gets drunk, the arteries, which can do so, dilate, the diseased ones cannot, and rupture may occur. A hysterical woman may have a genuine epileptic fit with post-epileptic coma, and she may have got drunk previous to either, and finally have been damaged.

Your preliminary actions are here the same as in the other groups. Clear a space round the patient, free his neck, listen to any history that is forthcoming, and if definitely pointing to poison act promptly on it. Do not forget that coma from alcohol is as much poisoning by that substance, and often as dangerous, as is poisoning by opium or any other drug.

Smell the Breath.—In the absence of a definite reliable history (either of an alcoholic bout or of only one glass given by anxious friends) the smell of alcohol is of equal importance with the smell of other poisonous substances. If a definite history is forthcoming an alcoholic smell must be judged by it.

Camphor, turpentine, carbolic acid, hydrocyanic acid, chloroform, æther, opium are the principal commoner poisons that will have imparted a distinctive odour to the breath. In diabetic coma the breath will be sweet like apples. Lately, on entering a patient's bedroom, I thought I had gone by mistake into an apple storeroom. Occasionally patients in uræmic coma are said to smell of ammonia.

Note the Face as Before.—If it is pale and dry the case is quite likely to be only simple fainting, if there are no other distinctive symptoms; if it is pale and sweaty, with cyanosed lips, cerebral hæmorrhage or poisoning are most probable; if flushed brightly it may be alcohol or belladonna poisoning, or possibly fever; if the face be purple and bloated, this points almost conclusively to serious cerebral mischief, probably compression.

Next Proceed to Examine the Pupils.—In the other sections we have already considered the broad outlines of the conclusions to be drawn from the condition of the pupils. We may here finally tabulate the possible conditions, with a few remarks on their special influence on final diagnosis.

The pupils may be—

As regards Reaction to Light.	As regards Absolute Size.	As regards Relative Size.
Active.	Dilated.	Equal.
Sluggish.	Normal.	Unequal.
Totally inactive.	Contracted.	

And finally either one or both may be irregular in outline.

Conditions in which the Pupils are still Active.—This is only likely to be the case when the condition of the brain causing the unconsciousness is either of a temporary character or in a very early stage. Inasmuch as we can but rarely see the patient in the latter condition, it is almost a fair clinical deduction to say that still very active pupils indicate no immediate danger. Thus they will be found in malingerers, in simple fainting (then indicative of returning consciousness), in hysterical and cataleptic conditions, and in the later stages of post-epileptic coma that is not going to prove fatal. The malingerer may be aware that he can, by stimulating the palm, dilate the pupil on that side, but this is such a temporary phenomenon as to cause little or no difficulty. In poisoning the pupils are frequently unaffected in the early stages, but this can hardly occur when absorption has so far advanced as to cause coma. The absolute size of active pupils will depend upon the amount of light in the examination room

Conditions in which the Pupils are Sluggish

This occurs in—

General paralysis of insane.	They will probably also be unequal, and behave otherwise in a bizarre manner, possibly dilating with light.
Uræmia.	May be small, natural in size, or dilated.
Disseminated sclerosis.	In any condition, like uræmia.
Early stages of apoplexy.	Will probably cease to act before the examination is completed.

Conditions in which the Pupil ceases to React to Light at all

The first and most important deduction from this state of the pupils (if we can exclude a glass eye and previous total blindness) is that the condition of brain underlying it is very serious, and even likely to prove fatal; it marks the dividing line between coma and stupor.

The commonest examples are:—

Pupils very small.	Opium poisoning (very rarely dilated). Pontine hæmorrhage (also often unequal). Possibly in general paralysis of the insane, or in tabes dorsalis.
Pupils large and equal.	Belladonna (and its allies) poisoning, and also in the later stages of most poisons, especially if

asphyxia is or has been a prominent feature. In connection with the large pupils of alcoholic poisoning it is worth while to note that even in a condition of coma due to this cause the pupils will still react to powerful peripheral stimuli, such as pinching the patient or pulling his hair forcibly. I know of no other grave condition of which this can be said.

Pupils unequal. Frequent in one-sided pontine lesions. Also in one-sided considerable increase in intracranial pressure, as from hæmorrhage.

In general paralysis, too, it may continue during a final coma if previously present.

After the examination of the eyes look for localised paralyses anywhere. Is the face reasonably symmetrical? Are the eyes in a natural axis or markedly deviating? Do the arms or the legs drop equally powerlessly when raised?

A localised paralysis of this sort is almost pathognomonic of coarse cerebral lesion, but of course in deep coma the point is often difficult to appreciate.

The next point is the respiration. If this is stertorous the probabilities are in favour of a cerebral lesion; but one must not forget that it may become stertorous in every form of dangerous coma.

Then the pulse. If it be small, compressible, and slow, it is very likely to be opium poisoning (occasionally opium produces a very rapid pulse) or pontine lesion. If it be small and thready and rapid, no conclusion can be drawn, as this pulse is met with in so many conditions of coma where death threatens. A slow, labouring pulse, especially with a thickened artery, is suggestive of cerebral hæmorrhage, or post-epileptic coma.

Now draw off some of the urine. If sugar be present, diabetic coma is rendered probable though it occurs when anything presses on the fourth ventricle; *e.g.* I have found it in cerebellar hæmorrhage. If it is uræmic coma, there is likely to be albumen, or possibly blood, or both, and the urine will be of low specific gravity; this latter is especially important, as the urine of cirrhotic kidney often enough contains no albumen, or such a faint trace as to be easily overlooked. A cirrhotic kidney, too, renders hæmorrhage very likely.

After noting the foregoing points, try how far the patient is capable of being roused; the danger increases *pari passu* with the depth of

the coma. In uræmia, even when likely to prove fatal, some signs of consciousness can usually be obtained by pretty forcible stimulation; this forms a valuable—not conclusive—distinction between uræmia and the coma of cerebral hæmorrhage.

Lastly, take the temperature. This may give some information; it will be very high in sunstroke or other forms of heat apoplexy, and gives instantly the best guide to treatment. It almost certainly will be raised in the comatose condition of general paralytics, or of inflammatory conditions, and hence will be of great use in separating these from hæmorrhage, uræmia, etc., in which the temperature (at the time we are considering) is invariably subnormal.

In conclusion, I must again repeat that no one of these indications is sufficient by itself; all must be considered and a balance struck. The history may be absolutely conclusive, and now and again an individual feature (*e.g.* conjugate deviation) may be so marked and obtrusive as to outweigh everything; but whatever conclusion we finally arrive at, we must NEVER LEAVE A COMATOSE PATIENT WITHOUT EFFICIENT SUPERVISION; HE MUST ALWAYS BE WATCHED CAREFULLY TILL A SUFFICIENT TIME HAS ELAPSED TO ALLOW OF AN ALCOHOLIC COMPLICATION HAVING PASSED OFF.

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THE END

DEFECTIVE EYESIGHT:

The Principles of Its Relief by Glasses.

BY

D. B. ST. JOHN ROOSA, M.D., LL.D.,

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